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(57) Abstract: The present invention relates to human secreted polypeptides, and isolated nucleic acid molecules encoding said polypeptides, useful for diagnosing and treating hematopoietic and hematologic diseases, disorders, and/or conditions related thereto. Antibodies that bind these polypeptides are also encompassed by the present invention. Also encompassed by the invention are vectors, host cells, and recombinant and synthetic methods for producing said polynucleotides, polypeptides, and/or antibodies. The invention further encompasses screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further encompasses methods and compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

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Human Secreted Proteins

Field of the Invention

The present invention relates to human secreted proteins/polypeptides, and isolated nucleic acid molecules encoding said proteins/polypeptides, useful for detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders. Antibodies that bind these polypeptides are also encompassed by the present invention. Also encompassed by the invention are vectors, host cells, and recombinant and synthetic methods for producing said polynucleotides, polypeptides, and/or antibodies. The invention further encompasses screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further encompasses methods and compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

Background of the Invention

Blood is composed of a fluid component, plasma, in which are suspended red blood cells, white blood cells, and platelets. This suspension, circulating through the cardiovascular system, forms the basis of the immune system, provides all of the body's tissues with oxygen and nutrients, and removes carbon dioxide and other metabolic byproducts for excretion.

Immune cells, red blood cells, and platelets, are derived from common precursor stem cells and develop through a process known as hematopoiesis. During fetal life hematopoiesis occurs in the liver and spleen, but in the adult, hematopoiesis occurs primarily in the bone marrow and thymus. The stem cells from which all blood cells are derived proliferate and differentiate into the various blood cell lineages, (e.g., lymphoid cells (B or T cells), myeloid cells (basophils, eosinophils, neutrophils, macrophages, mast cells), thrombocytes (platelets), or erythrocytes (red blood cells)) in response to cytokines and other signals received from cells (e.g., stromal cells) in the bone marrow microenvironment. Many of the cytokines that promote the growth and differentiation of hematopoietic stem cells are known as "colony stimulating factors". For example, interleukin-3 (IL-3, and also known as multi-colony stimulating factor) and granulocyte macrophage colony stimulating factor (GM-CSF), which are released by activated macrophages and T cells, stimulate the production of macrophages and granulocytes (myelopoiesis). Stem cell factor (SCF, c-kit ligand) is a growth factor for primitive lymphoid and myeloid hematopoietic

bone marrow progenitor cells expressing the early cell surface marker CD34. Other hematopoietic cytokines/growth factors include, but are not limited to macrophage colony stimulating factor (M-CSF), granulocyte colony stimulating factor (G-CSF), and erythropoietin (EPO). Interleukins-1, 6, and 7 have also been shown to function as hematopoietic growth factors/cytokines. Deficiencies in the quantities of mature red or white blood cells, either as a result of insufficient production or excessive destruction, may result in anemias and/or immunodeficiencies.

In addition to the cellular component of the blood, there are a remarkable variety of soluble blood-borne proteins that serve important physiological functions. Descriptions of some of the functional classes of blood proteins, along with representative members of these classes, are given below.

Coagulation factors

Immunoproteins

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The formation of insoluble protein aggregates at the site of vascular injury or inflammation, termed coagulation, is the result of multiple interacting coagulation factors (Dahlback, B., Lancet 355:1627-32). This cascade of interdependent proteins (including Factors V, VIII, IX, X XI, and XII) results in the production of the protease, thrombin. Thrombin converts blood-soluble fibrinogen into fibrin, which polymerizes into insoluble clots that are stabilized by the activity of Factor XIII. This process is balanced by the activity of coagulation inhibitors such as antithrombin III, heparin cofactor II, Protein C and Protein S. Imbalance between pro-clotting factors and coagulation inhibitors leads to potentially serious medical conditions, including improper wound healing and the bleeding disorders hemophilia A and B, as well as excessive clotting disorders such as thrombosis (e.g. cerebral, coronary, and placental), pulmonary embolus, stroke, and coronary artery disease. For a more extensive review see Triplett, D., Clin Chem 46:1260-9.

Blood plasma contains a number of proteins that contribute to the immune response. Immunoglobulin antibodies are glycoproteins with similar structural domains, which bind to specific antigenic invaders and trigger other components of the immune system. The complement cascade, a network of about 20 interacting proteins, is activated by antigen-antibody complexes and results in the lysis of infected cells, as well as other important immune functions. Immunoproteins are important tools for the diagnosis and treatment of infection, cancer, and other disorders. For more detailed discussion of immunoproteins see Meri, J. and Jarva, H., *Vox Sang* 74 suppl. 2:291-302 and Chapter 23 of Molecular Biology of the Cell, 3rd Edition, edited by Alberts, B. et al.

35 Hormones

The blood serves as a major vehicle for hormones and other secreted signaling molecules that act at a site distant to their release. A number of peptide hormones function as regulators of

homeostatic processes. For example, parathyroid hormone and calcitonin oppositely regulate serum levels of calcium. Blood-borne peptide hormones that regulate carbohydrate metabolism include insulin, glucagon, and adrenocorticotropin hormone. Vasopressin, angiotensin, and bradykinin are hormones that modulate vasodilation and blood pressure. Follicle-stimulating hormone and leutinizing hormone play important roles in both male and female reproductive functions. Dysfunction of these hormones can lead to a wide spectrum of disorders, including osteoporosis, diabetes, psychiatric disorders, hypoglycemia, obesity, infertility, as well as hypoand hypertension.

10 Cytokines

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Cytokines are a class of circulating proteins that act primarily as intercellular signaling molecules regulating hematopoiesis, angiogenesis, and immune system functions. One subgroup of cytokines, the hematopoietins, regulates hematopoietic stem cell differentiation to maintain the proper number and proportions of each blood cell type. For example, the production of erythrocytes is stimulated by the release of erythropoietin from the kidneys in response to decreased blood oxygen levels. Similarly, thrombopoietin stimulates the proliferation and differentiation of megakaryocytes, leading to increased platelet production. Another cytokine subgroup, the chemokines, is secreted by cells of the immune system, and act to coordinate the immune response to an invading antigen. This is a large and diverse class of proteins, and includes RANTES, eotaxin, lymphotactin, MIP-1, and the interleukins. Many of these polypeptides have uses in the diagnosis and treatment of immunological disorders and infection (Holldack, J. et al., Med Ped Oncol Suppl 2:2-9; Chapter 23, Immunology, edited by Elgert, K.).

Carrier Proteins

A number of soluble proteins found in blood function as carriers of other molecules such as nutrients and waste products. Carrier proteins can also bind exogenously delivered drugs and influence pharmacokinetic properties such as serum half-life and tissue adsorption. Serum albumin, comprising about half of the protein found in blood plasma, regulates osmotic pressure of blood, as well as binds many bioactive molecules. Transferrin is a blood carrier protein that regulates iron levels, while ceruloplasmin regulates copper levels.

Thus there exists a clear need for novel polynucleotides and polypeptides (as well as antibodies, agonists, and antagonists) useful in diagnostic and therapeutic methods for detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders; such as, for example, leukemias, lymphomas, hemophilias, anemias, immunodeficiency disorders (including AIDS), amongst many other conditions. See, e.g., "Blood Related Disorders" and "Immune Activity" sections, infra.

Summary of the Invention

The present invention encompasses human secreted proteins/polypeptides, and isolated nucleic acid molecules encoding said proteins/polypeptides, useful for detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders. Antibodies that bind these polypeptides are also encompassed by the present invention; as are vectors, host cells, and recombinant and synthetic methods for producing said polynucleotides, polypeptides, and/or antibodies. The invention further encompasses screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention also encompasses methods and compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

Detailed Description

Polynucleotides and Polypeptides of the Invention

Description of Table 1A

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Table 1A summarizes information concerning certain polypnucleotides and polypeptides of the invention. The first column provides the gene number in the application for each clone identifier. The second column provides a unique clone identifier, "Clone ID:", for a cDNA clone related to each contig sequence disclosed in Table 1A. Third column, the cDNA Clones identified in the second column were deposited as indicated in the third column (i.e. by ATCC Deposit No:Z and deposit date). Some of the deposits contain multiple different clones corresponding to the same gene. In the fourth column, "Vector" refers to the type of vector contained in the corresponding cDNA Clone identified in the second column. In the fifth column, the nucleotide sequence identified as "NT SEQ ID NO:X" was assembled from partially homologous ("overlapping") sequences obtained from the corresponding cDNA clone identified in the second column and, in some cases, from additional related cDNA clones. The overlapping sequences were assembled into a single contiguous sequence of high redundancy (usually three to five overlapping sequences at each nucleotide position), resulting in a final sequence identified as SEQ ID NO:X. In the sixth column, "Total NT Seq." refers to the total number of nucleotides in the contig sequence identified as SEO ID NO:X." The deposited clone may contain all or most of these sequences, reflected by the nucleotide position indicated as "5' NT of Clone Seq." (seventh column) and the "3" NT of Clone Seq." (eighth column) of SEQ ID NO:X. In the ninth column,

the nucleotide position of SEQ ID NO:X of the putative start codon (methionine) is identified as "5' NT of Start Codon." Similarly, in column ten, the nucleotide position of SEQ ID NO:X of the predicted signal sequence is identified as "5' NT of First AA of Signal Pep." In the eleventh column, the translated amino acid sequence, beginning with the methionine, is identified as "AA SEQ ID NO:Y," although other reading frames can also be routinely translated using known molecular biology techniques. The polypeptides produced by these alternative open reading frames are specifically contemplated by the present invention.

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In the twelfth and thirteenth columns of Table 1A, the first and last amino acid position of SEQ ID NO:Y of the predicted signal peptide is identified as "First AA of Sig Pep" and "Last AA of Sig Pep." In the fourteenth column, the predicted first amino acid position of SEQ ID NO:Y of the secreted portion is identified as "Predicted First AA of Secreted Portion". The amino acid position of SEQ ID NO:Y of the last amino acid encoded by the open reading frame is identified in the fifteenth column as "Last AA of ORF".

SEQ ID NO:X (where X may be any of the polynucleotide sequences disclosed in the sequence listing) and the translated SEQ ID NO:Y (where Y may be any of the polypeptide sequences disclosed in the sequence listing) are sufficiently accurate and otherwise suitable for a variety of uses well known in the art and described further below. For instance, SEQ ID NO:X is useful for designing nucleic acid hybridization probes that will detect nucleic acid sequences contained in SEQ ID NO:X or the cDNA contained in the deposited clone. These probes will also hybridize to nucleic acid molecules in biological samples, thereby enabling a variety of forensic and diagnostic methods of the invention. Similarly, polypeptides identified from SEQ ID NO:Y may be used, for example, to generate antibodies which bind specifically to proteins containing the polypeptides and the secreted proteins encoded by the cDNA clones identified in Table 1A and/or elsewhere herein

Nevertheless, DNA sequences generated by sequencing reactions can contain sequencing errors. The errors exist as misidentified nucleotides, or as insertions or deletions of nucleotides in the generated DNA sequence. The erroneously inserted or deleted nucleotides cause frame shifts in the reading frames of the predicted amino acid sequence. In these cases, the predicted amino acid sequence diverges from the actual amino acid sequence, even though the generated DNA sequence may be greater than 99.9% identical to the actual DNA sequence (for example, one base insertion or deletion in an open reading frame of over 1000 bases).

Accordingly, for those applications requiring precision in the nucleotide sequence or the amino acid sequence, the present invention provides not only the generated nucleotide sequence identified as SEQ ID NO:X, and the predicted translated amino acid sequence identified as SEQ ID NO:Y, but also a sample of plasmid DNA containing a human cDNA of the invention deposited with the ATCC, as set forth in Table 1A. The nucleotide sequence of each deposited

plasmid can readily be determined by sequencing the deposited plasmid in accordance with known methods

The predicted amino acid sequence can then be verified from such deposits. Moreover, the amino acid sequence of the protein encoded by a particular plasmid can also be directly determined by peptide sequencing or by expressing the protein in a suitable host cell containing the deposited human cDNA, collecting the protein, and determining its sequence.

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Also provided in Table 1A is the name of the vector which contains the cDNA plasmid. Each vector is routinely used in the art. The following additional information is provided for convenience.

Vectors Lambda Zap (U.S. Patent Nos. 5,128,256 and 5,286,636), Uni-Zap XR (U.S. Patent Nos. 5,128, 256 and 5,286,636), Zap Express (U.S. Patent Nos. 5,128,256 and 5,286,636), pBluescript (pBS) (Short, J. M. et al., Nucleic Acids Res. 16:7583-7600 (1988); Alting-Mees, M. A. and Short, J. M., Nucleic Acids Res. 17:9494 (1989)) and pBK (Alting-Mees, M. A. et al., Strategies 5:58-61 (1992)) are commercially available from Stratagene Cloning Systems, Inc., 11011 N. Torrey Pines Road, La Jolla, CA, 92037. pBS contains an ampicillin resistance gene and pBK contains a neomycin resistance gene. Phagemid pBS may be excised from the Lambda Zap and Uni-Zap XR vectors, and phagemid pBK may be excised from the Zap Express vector. Both phagemids may be transformed into E. coli strain XL-1 Blue, also available from Stratagene

Vectors pSport1, pCMVSport 1.0, pCMVSport 2.0 and pCMVSport 3.0, were obtained from Life Technologies, Inc., P. O. Box 6009, Gaithersburg, MD 20897. All Sport vectors contain an ampicillin resistance gene and may be transformed into *E. coli* strain DH10B, also available from Life Technologies. See, for instance, Gruber, C. E., et al., *Focus* 15:59 (1993). Vector lafmid BA (Bento Soares, Columbia University, New York, NY) contains an ampicillin resistance gene and can be transformed into *E. coli* strain XL-1 Blue. Vector pCR[®]2.1, which is available from Invitrogen, 1600 Faraday Avenue, Carlsbad, CA 92008, contains an ampicillin resistance gene and may be transformed into *E. coli* strain DH10B, available from Life Technologies. See, for instance, Clark, J. M., *Nuc. Acids Res.* 16:9677-9686 (1988) and Mead, D. et al., Bio/Technology 9: (1991).

The present invention also relates to the genes corresponding to SEQ ID NO:X, SEQ ID NO:Y, and/or a deposited cDNA (cDNA Clone ID). The corresponding gene can be isolated in accordance with known methods using the sequence information disclosed herein. Such methods include, but are not limited to, preparing probes or primers from the disclosed sequence and identifying or amplifying the corresponding gene from appropriate sources of genomic material.

Also provided in the present invention are allelic variants, orthologs, and/or species homologs. Procedures known in the art can be used to obtain full-length genes, allelic variants, splice variants, full-length coding portions, orthologs, and/or species homologs of genes corresponding to SEQ ID NO:X and SEQ ID NO:Y using information from the sequences

disclosed herein or the clones deposited with the ATCC. For example, allelic variants and/or species homologs may be isolated and identified by making suitable probes or primers from the sequences provided herein and screening a suitable nucleic acid source for allelic variants and/or the desired homologue.

The present invention provides a polynucleotide comprising, or alternatively consisting of, the nucleic acid sequence of SEQ ID NO:X and/or a cDNA contained in ATCC Deposit No.Z. The present invention also provides a polypeptide comprising, or alternatively, consisting of, the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X, and/or a polypeptide encoded by a cDNA contained in ATCC deposit No.Z. Polynucleotides encoding a polypeptide comprising, or alternatively consisting of the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X and/or a polypeptide encoded by the cDNA contained in ATCC Deposit No.Z, are also encompassed by the invention. The present invention further encompasses a polynucleotide comprising, or alternatively consisting of the complement of the nucleic acid sequence of SEQ ID NO:X, and/or the complement of the coding strand of the cDNA contained in ATCC Deposit No.Z.

Description of Table 1B (Comprised of Tables 1B.1 and 1B.2)

Table 1B.1 and Table 1B.2 summarize some of the polynucleotides encompassed by the invention (including cDNA clones related to the sequences (Clone ID:), contig sequences (contig identifier (Contig ID:) and contig nucleotide sequence identifiers (SEQ ID NO:X)) and further summarizes certain characteristics of these polynucleotides and the polypeptides encoded thereby. The first column of Tables 1B.1 and 1B.2 provide the gene numbers in the application for each clone identifier. The second column of Tables 1B.1 and 1B.2 provide unique clone identifiers, "Clone ID:", for cDNA clones related to each contig sequence disclosed in Table 1A and/or Table 1B. The third column of Tables 1B.1 and 1B.2 provide unique contig identifiers, "Contig ID:" for each of the contig sequences disclosed in these tables. The fourth column of Tables 1B.1 and 1B.2 provide the sequence identifiers, "SEQ ID NO:X", for each of the contig sequences disclosed in Table 1A and/or 1B.

Table 1B.1

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The fifth column of Table 1B.1, "ORF (From-To)", provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence of SEQ ID NO:X that delineates the preferred open reading frame (ORF) that encodes the amino acid sequence shown in the sequence listing and referenced in Table 1B.1 as SEQ ID NO:Y (column 6). Column 7 of Table 1B.1 lists residues comprising predicted epitopes contained in the polypeptides encoded by each of the preferred ORFs (SEQ ID NO:Y). Identification of potential immunogenic regions was performed according to the method of Jameson and Wolf (CABIOS, 4; 181-186 (1988)); specifically, the Genetics Computer Group (GCG) implementation of this algorithm, embodied in

the program PEPTIDESTRUCTURE (Wisconsin Package v10.0, Genetics Computer Group (GCG), Madison, Wisc.). This method returns a measure of the probability that a given residue is found on the surface of the protein. Regions where the antigenic index score is greater than 0.9 over at least 6 amino acids are indicated in Table 1B.1 as "Predicted Epitopes". In particular embodiments, polypeptides of the invention comprise, or alternatively consist of, one, two, three, four, five or more of the predicted epitopes described in Table 1B.1. It will be appreciated that depending on the analytical criteria used to predict antigenic determinants, the exact address of the determinant may vary slightly. Column 8 of Table 1B.1 ("Cytologic Band") provides the chromosomal location of polynucleotides corresponding to SEQ ID NO:X. Chromosomal location was determined by finding exact matches to EST and cDNA sequences contained in the NCBI (National Center for Biotechnology Information) UniGene database. Given a presumptive chromosomal location, disease locus association was determined by comparison with the Morbid Map, derived from Online Mendelian Inheritance in Man (Online Mendelian Inheritance in Man, OMIMTM. McKusick-Nathans Institute for Genetic Medicine, Johns Hopkins University (Baltimore, MD) and National Center for Biotechnology Information, National Library of Medicine (Bethesda, MD) 2000. World Wide Web URL: http://www.ncbi.nlm.nih.gov/omim/). If the putative chromosomal location of the Query overlaps with the chromosomal location of a Morbid Map entry, an OMIM identification number is disclosed in Table 1B.1, column 9 labeled "OMIM Disease Reference(s)". A key to the OMIM reference identification numbers is provided in Table 5.

Table 1B.2

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Column 5 of Table 1B.2, "Tissue Distribution" shows the expression profile of tissue, cells, and/or cell line libraries which express the polynucleotides of the invention. The first code number shown in Table 1B.2 column 5 (preceding the colon), represents the tissue/cell source identifier code corresponding to the key provided in Table 4. Expression of these polynucleotides was not observed in the other tissues and/or cell libraries tested. The second number in column 5 (following the colon), represents the number of times a sequence corresponding to the reference polynucleotide sequence (e.g., SEQ ID NO:X) was identified in the corresponding tissue/cell source. Those tissue/cell source identifier codes in which the first two letters are "AR" designate information generated using DNA array technology. Utilizing this technology, cDNAs were amplified by PCR and then transferred, in duplicate, onto the array. Gene expression was assayed through hybridization of first strand cDNA probes to the DNA array. cDNA probes were generated from total RNA extracted from a variety of different tissues and cell lines. Probe synthesis was performed in the presence of ³³P dCTP, using oligo(dT) to prime reverse transcription. After hybridization, high stringency washing conditions were employed to remove non-specific hybrids from the array. The remaining signal, emanating from each gene target, was measured using a Phosphorimager. Gene expression was reported as Phosphor Stimulating Luminescence (PSL)

which reflects the level of phosphor signal generated from the probe hybridized to each of the gene targets represented on the array. A local background signal subtraction was performed before the total signal generated from each array was used to normalize gene expression between the different hybridizations. The value presented after "[array code]:" represents the mean of the duplicate values, following background subtraction and probe normalization. One of skill in the art could routinely use this information to identify normal and/or diseased tissue(s) which show a predominant expression pattern of the corresponding polynucleotide of the invention or to identify polynucleotides which show predominant and/or specific tissue and/or cell expression.

10 Description of Table 1C

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Table 1C summarizes additional polynucleotides encompassed by the invention (including cDNA clones related to the sequences (Clone ID:), contig sequences (contig identifier (Contig ID:) contig nucleotide sequence identifiers (SEQ ID NO:X)), and genomic sequences (SEQ ID NO:B). The first column provides a unique clone identifier, "Clone ID:", for a cDNA clone related to each contig sequence. The second column provides the sequence identifier, "SEQ ID NO:X", for each contig sequence. The third column provides a unique contig identifier, "Contig ID:" for each contig sequence. The fourth column, provides a BAC identifier "BAC ID NO:A" for the BAC clone referenced in the corresponding row of the table. The fifth column provides the nucleotide sequence identifier, "SEQ ID NO:B" for a fragment of the BAC clone identified in column four of the corresponding row of the table. The sixth column, "Exon From-To", provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence of SEQ ID NO:B which delineate certain polynucleotides of the invention that are also exemplary members of polynucleotide sequences that encode polypeptides of the invention (e.g., polypeptides containing amino acid sequences encoded by the polynucleotide sequences delineated in column six, and fragments and variants thereof).

Description of Table 1D

Table 1D: In preferred embodiments, the present invention encompasses a method of detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders; comprising administering to a patient in which such treatment, prevention, or amelioration is desired a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof) represented by Table 1A, Table 1B, and Table 1C, in an amount effective to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate the disease or disorder.

As indicated in Table 1D, the polynucleotides, polypeptides, agonists, or antagonists of the present invention (including antibodies) can be used in assays to test for one or more biological activities. If these polynucleotides and polypeptides do exhibit activity in a particular assay, it is

likely that these molecules may be involved in the diseases associated with the biological activity. Thus, the polynucleotides or polypeptides, or agonists or antagonists thereof (including antibodies) could be used to treat the associated disease.

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Table 1D provides information related to biological activities for polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof). Table 1D also provides information related to assays which may be used to test polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof) for the corresponding biological activities. The first column ("Gene No.") provides the gene number in the application for each clone identifier. The second column ("cDNA Clone ID:") provides the unique clone identifier for each clone as previously described and indicated in Tables 1A, 1B, and 1C. The third column ("AA SEQ ID NO:Y") indicates the Sequence Listing SEQ ID Number for polypeptide sequences encoded by the corresponding cDNA clones (also as indicated in Tables 1A, 1B, and 2). The fourth column ("Biological Activity") indicates a biological activity corresponding to the indicated polypeptides (or polynucleotides encoding said polypeptides). The fifth column ("Exemplary Activity Assay") further describes the corresponding biological activity and provides information pertaining to the various types of assays which may be performed to test, demonstrate, or quantify the corresponding biological activity. Table 1D describes the use of FMAT technology, inter alia, for testing or demonstrating various biological activities. Fluorometric microvolume assay technology (FMAT) is a fluorescence-based system which provides a means to perform nonradioactive cell- and bead-based assays to detect activation of cell signal transduction pathways. This technology was designed specifically for ligand binding and immunological assays. Using this technology, fluorescent cells or beads at the bottom of the well are detected as localized areas of concentrated fluorescence using a data processing system. Unbound flurophore comprising the background signal is ignored, allowing for a wide variety of homogeneous assays. FMAT technology may be used for peptide ligand binding assays, immunofluorescence, apoptosis, cytotoxicity, and bead-based immunocapture assays. Miraglia S et. al., "Homogeneous cell and bead based assays for highthroughput screening using flourometric microvolume assay technology," Journal of Biomolecular Screening; 4:193-204 (1999). In particular, FMAT technology may be used to test, confirm, and/or identify the ability of polypeptides (including polypeptide fragments and variants) to activate signal transduction pathways. For example, FMAT technology may be used to test, confirm, and/or identify the ability of polypeptides to upregulate production of immunomodulatory proteins (such as, for example, interleukins, GM-CSF, Rantes, and Tumor Necrosis factors, as well as other cellular regulators (e.g. insulin)).

Table 1D also describes the use of kinase assays for testing, demonstrating, or quantifying biological activity. In this regard, the phosphorylation and de-phosphorylation of specific amino acid residues (e.g. Tyrosine, Serine, Threonine) on cell-signal transduction proteins provides a fast,

reversible means for activation and de-activation of cellular signal transduction pathways. Moreover, cell signal transduction via phosphorylation/de-phosphorylation is crucial to the regulation of a wide variety of cellular processes (e.g. proliferation, differentiation, migration, apoptosis, etc.). Accordingly, kinase assays provide a powerful tool useful for testing, confirming, and/or identifying polypeptides (including polypeptide fragments and variants) that mediate cell signal transduction events via protein phosphorylation. See e.g., Forrer, P., Tamaskovic R., and Jaussi, R. "Enzyme-Linked Immunosorbent Assay for Measurement of JNK, ERK, and p38 Kinase Activities" Biol. Chem. 379(8-9): 1101-1110 (1998).

10 Description of Table 1E

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Polynucleotides encoding polypeptides of the present invention can be used in assays to test for one or more biological activities. One such biological activity which may be tested includes the ability of polynucleotides and polypeptides of the invention to stimulate up-regulation or down-regulation of expression of particular genes and proteins. Hence, if polynucleotides and polypeptides of the present invention exhibit activity in altering particular gene and protein expression patterns, it is likely that these polynucleotides and polypeptides of the present invention may be involved in, or capable of effecting changes in, diseases associated with the altered gene and protein expression profiles. Hence, polynucleotides, polypeptides, or antibodies of the present invention could be used to treat said associated diseases.

TaqMan® assays may be performed to assess the ability of polynucleotides (and polypeptides they encode) to alter the expression pattern of particular "target" genes. TaqMan® reactions are performed to evaluate the ability of a test agent to induce or repress expression of specific genes in different cell types. TaqMan® gene expression quantification assays ("TaqMan® assays") are well known to, and routinely performed by, those of ordinary skill in the art. TaqMan® assays are performed in a two step reverse transcription / polymerase chain reaction (RT-PCR). In the first (RT) step, cDNA is reverse transcribed from total RNA samples using random hexamer primers. In the second (PCR) step, PCR products are synthesized from the cDNA using gene specific primers.

To quantify gene expression the Taqman® PCR reaction exploits the 5' nuclease activity of AmpliTaq Gold® DNA Polymerase to cleave a Taqman® probe (distinct from the primers) during PCR. The Taqman® probe contains a reporter dye at the 5'-end of the probe and a quencher dye at the 3' end of the probe. When the probe is intact, the proximity of the reporter dye to the quencher dye results in suppression of the reporter fluorescence. During PCR, if the target of interest is present, the probe specifically anneals between the forward and reverse primer sites. AmpliTaq Fold DNA Polymerase then cleaves the probe between the reporter and quencher when the probe hybridizes to the target, resulting in increased fluorescence of the reporter (see

Figure 2). Accumulation of PCR products is detected directly by monitoring the increase in fluorescence of the reporter dye.

After the probe fragments are displaced from the target, polymerization of the strand continues. The 3'-end of the probe is blocked to prevent extension of the probe during PCR. This process occurs in every cycle and does not interfere with the exponential accumulation of product. The increase in fluorescence signal is detected only if the target sequence is complementary to the probe and is amplified during PCR. Because of these requirements, any nonspecific amplification is not detected.

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For test sample preparation, vector controls or constructs containing the coding sequence for the gene of interest are transfected into cells, such as for example 293T cells, and supernatants collected after 48 hours. For cell treatment and RNA isolation, multiple primary human cells or human cell lines are used; such cells may include but are not limited to, Normal Human Dermal Fibroblasts, Aortic Smooth Muscle, Human Umbilical Vein Endothelial Cells, HepG2, Daudi, Jurkat, U937, Caco, and THP-1 cell lines. Cells are plated in growth media and growth is arrested by culturing without media change for 3 days, or by switching cells to low serum media and incubating overnight. Cells are treated for 1, 6, or 24 hours with either vector control supernatant or sample supernatant (or purified/partially purified protein preparations in buffer). Total RNA is isolated; for example, by using Trizol extraction or by using the Ambion RNAqueous(TM)-4PCR RNA isolation system. Expression levels of multiple genes are analyzed using TAQMAN, and expression in the test sample is compared to control vector samples to identify genes induced or repressed. Each of the above described techniques are well known to, and routinely performed by, those of ordinary skill in the art.

Table 1E indicates particular disease classes and preferred indications for which polynucleotides, polypeptides, or antibodies of the present invention may be used in detecting, diagnosing, preventing, treating and/or ameliorating said diseases and disorders based on "target" gene expression patterns which may be up- or down-regulated by polynucleotides (and the encoded polypeptides) corresponding to each indicated cDNA Clone ID (shown in Table 1E, Column 2).

Thus, in preferred embodiments, the present invention encompasses a method of detecting, diagnosing, preventing, treating, and/or ameliorating a disease or disorder listed in the "Disease Class" and/or "Preferred Indication" columns of Table 1E; comprising administering to a patient in which such detection, diagnosis, prevention, or treatment is desired a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof) in an amount effective to detect, diagnose, prevent, treat, or ameliorate the disease or disorder. The first and second columns of Table 1D show the "Gene No." and "cDNA Clone ID No.", respectively, indicating certain nucleic acids and proteins (or antibodies against the same) of the invention (including polynucleotide, polypeptide, and antibody fragments or variants thereof) that may be used in detecting, diagnosing,

preventing, treating, or ameliorating the disease(s) or disorder(s) indicated in the corresponding row in the "Disease Class" or "Preferred Indication" Columns of Table 1E.

In another embodiment, the present invention also encompasses methods of detecting, diagnosing, preventing, treating, or ameliorating a disease or disorder listed in the "Disease Class" or "Preferred Indication" Columns of Table 1E; comprising administering to a patient combinations of the proteins, nucleic acids, or antibodies of the invention (or fragments or variants thereof), sharing similar indications as shown in the corresponding rows in the "Disease Class" or "Preferred Indication" Columns of Table 1E.

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The "Disease Class" Column of Table 1E provides a categorized descriptive heading for diseases, disorders, and/or conditions (more fully described below) that may be detected, diagnosed, prevented, treated, or ameliorated by a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof).

The "Preferred Indication" Column of Table 1E describes diseases, disorders, and/or conditions that may be detected, diagnosed, prevented, treated, or ameliorated by a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof).

The "Cell Line" and "Exemplary Targets" Columns of Table 1E indicate particular cell lines and target genes, respectively, which may show altered gene expression patterns (i.e., up- or down-regulation of the indicated target gene) in Taqman assays, performed as described above, utilizing polynucleotides of the cDNA Clone ID shown in the corresponding row. Alteration of expression patterns of the indicated "Exemplary Target" genes is correlated with a particular "Disease Class" and/or "Preferred Indication" as shown in the corresponding row under the respective column headings.

The "Exemplary Accessions" Column indicates GenBank Accessions (available online through the National Center for Biotechnology Information (NCBI) at http://www.ncbi.nlm.nih.gov/) which correspond to the "Exemplary Targets" shown in the adjacent row.

The recitation of "Cancer" in the "Disease Class" Column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof) may be used for example, to detect, diagnose, prevent, treat, and/or ameliorate neoplastic diseases and/or disorders (e.g., leukemias, cancers, etc., as described below under "Hyperproliferative Disorders").

The recitation of "Immune" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof), may be used for example, to detect, diagnose, prevent, treat, and/or ameliorate diseases and/or disorders relating to neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune

Activity" "Cardiovascular Disorders" and/or "Blood-Related Disorders"), and infections (e.g., as described below under "Infectious Disease").

The recitation of "Angiogenesis" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof), may be used for example, to detect, diagnose, treat, prevent, and/or ameliorate diseases and/or disorders relating to neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), diseases and/or disorders of the cardiovascular system (e.g., as described below under "Cardiovascular Disorders"), diseases and/or disorders involving cellular and genetic abnormalities (e.g., as described below under "Diseases at the Cellular Level"), diseases and/or disorders involving angiogenesis (e.g., as described below under "Anti-Angiogenesis Activity"), to promote or inhibit cell or tissue regeneration (e.g., as described below under "Regeneration"), or to promote wound healing (e.g., as described below under "Wound Healing and Epithelial Cell Proliferation").

The recitation of "Diabetes" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof), may be used for example, to detect, diagnose, treat, prevent, and/or ameliorate diabetes (including diabetes mellitus types I and II), as well as diseases and/or disorders associated with, or consequential to, diabetes (e.g. as described below under "Endocrine Disorders," "Renal Disorders," and "Gastrointestinal Disorders").

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Description of Table 2

Table 2 summarizes homology and features of some of the polypeptides of the invention. The first column provides a unique clone identifier, "Clone ID:", corresponding to a cDNA clone disclosed in Table 1A or Table 1B. The second column provides the unique contig identifier, "Contig ID:" corresponding to contigs in Table 1B and allowing for correlation with the information in Table 1B. The third column provides the sequence identifier, "SEQ ID NO:X", for the contig polynucleotide sequence. The fourth column provides the analysis method by which the homology/identity disclosed in the Table was determined. Comparisons were made between polypeptides encoded by the polynucleotides of the invention and either a non-redundant protein database (herein referred to as "NR"), or a database of protein families (herein referred to as "PFAM") as further described below. The fifth column provides a description of the PFAM/NR hit having a significant match to a polypeptide of the invention. Column six provides the accession number of the PFAM/NR hit disclosed in the fifth column. Column seven, "Score/Percent Identity", provides a quality score or the percent identity, of the hit disclosed in columns five and six. Columns 8 and 9, "NT From" and "NT To" respectively, delineate the polynucleotides in "SEQ ID NO:X" that encode a polypeptide having a significant match to the PFAM/NR database as disclosed in the fifth and sixth columns. In specific embodiments

polypeptides of the invention comprise, or alternatively consist of, an amino acid sequence encoded by a polynucleotide in SEQ ID NO:X as delineated in columns 8 and 9, or fragments or variants thereof.

5 Description of Table 3

Table 3 provides polynucleotide sequences that may be disclaimed according to certain embodiments of the invention. The first column provides a unique clone identifier, "Clone ID", for a cDNA clone related to contig sequences disclosed in Table 1B. The second column provides the sequence identifier, "SEQ ID NO:X", for contig sequences disclosed in Table 1A and/or Table 1B. The third column provides the unique contig identifier, "Contig ID:", for contigs disclosed in Table 1B. The fourth column provides a unique integer 'a' where 'a' is any integer between 1 and the final nucleotide minus 15 of SEQ ID NO:X, and the fifth column provides a unique integer 'b' where 'b' is any integer between 15 and the final nucleotide of SEQ ID NO:X, where both a and b correspond to the positions of nucleotide residues shown in SEQ ID NO:X, and where b is greater than or equal to a + 14. For each of the polynucleotides shown as SEQ ID NO:X, the uniquely defined integers can be substituted into the general formula of a-b, and used to describe polynucleotides which may be preferably excluded from the invention. In certain embodiments, preferably excluded from the invention are at least one, two, three, four, five, ten, or more of the polynucleotide sequence(s) having the accession number(s) disclosed in the sixth column of this Table (including for example, published sequence in connection with a particular BAC clone). In further embodiments, preferably excluded from the invention are the specific polynucleotide sequence(s) contained in the clones corresponding to at least one, two, three, four, five, ten, or more of the available material having the accession numbers identified in the sixth column of this Table (including for example, the actual sequence contained in an identified BAC clone).

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Description of Table 4

Table 4 provides a key to the tissue/cell source identifier code disclosed in Table 1B.2, column 5. Column 1 of Table 4 provides the tissue/cell source identifier code disclosed in Table 1B.2, Column 5. Columns 2-5 provide a description of the tissue or cell source. Note that "Description" and "Tissue" sources (i.e. columns 2 and 3) having the prefix "a_" indicates organs, tissues, or cells derived from "adult" sources. Codes corresponding to diseased tissues are indicated in column 6 with the word "disease." The use of the word "disease" in column 6 is non-limiting. The tissue or cell source may be specific (e.g. a neoplasm), or may be disease-associated (e.g., a tissue sample from a normal portion of a diseased organ). Furthermore, tissues and/or cells lacking the "disease" designation may still be derived from sources directly or indirectly involved in a disease state or disorder, and therefore may have a further utility in that disease state or disorder. In numerous cases where the tissue/cell source is a library, column 7 identifies the vector

used to generate the library.

Description of Table 5

Table 5 provides a key to the OMIM reference identification numbers disclosed in Table 1B.1, column 9. OMIM reference identification numbers (Column 1) were derived from Online Mendelian Inheritance in Man (Online Mendelian Inheritance in Man, OMIM. McKusick-Nathans Institute for Genetic Medicine, Johns Hopkins University (Baltimore, MD) and National Center for Biotechnology Information, National Library of Medicine, (Bethesda, MD) 2000. World Wide Web URL: http://www.ncbi.nlm.nih.gov/omim/). Column 2 provides diseases associated with the cytologic band disclosed in Table 1B.1, column 8, as determined using the Morbid Map database.

Description of Table 6

Table 6 summarizes some of the ATCC Deposits, Deposit dates, and ATCC designation numbers of deposits made with the ATCC in connection with the present application. These deposits were made in addition to those described in the Table 1A.

Description of Table 7

Table 7 shows the cDNA libraries sequenced, and ATCC designation numbers and vector information relating to these cDNA libraries.

The first column shows the first four letters indicating the Library from which each library clone was derived. The second column indicates the catalogued tissue description for the corresponding libraries. The third column indicates the vector containing the corresponding clones. The fourth column shows the ATCC deposit designation for each library clone as indicated by the deposit information in Table 6.

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Definitions

The following definitions are provided to facilitate understanding of certain terms used throughout this specification.

In the present invention, "isolated" refers to material removed from its original environment (e.g., the natural environment if it is naturally occurring), and thus is altered "by the hand of man" from its natural state. For example, an isolated polynucleotide could be part of a vector or a composition of matter, or could be contained within a cell, and still be "isolated" because that vector, composition of matter, or particular cell is not the original environment of the polynucleotide. The term "isolated" does not refer to genomic or cDNA libraries, whole cell total or mRNA preparations, genomic DNA preparations (including those separated by electrophoresis and transferred onto blots), sheared whole cell genomic DNA preparations or other compositions

where the art demonstrates no distinguishing features of the polynucleotide/sequences of the present invention.

In the present invention, a "secreted" protein refers to those proteins capable of being directed to the ER, secretory vesicles, or the extracellular space as a result of a signal sequence, as well as those proteins released into the extracellular space without necessarily containing a signal sequence. If the secreted protein is released into the extracellular space, the secreted protein can undergo extracellular processing to produce a "mature" protein. Release into the extracellular space can occur by many mechanisms, including exocytosis and proteolytic cleavage.

As used herein, a "polynucleotide" refers to a molecule having a nucleic acid sequence encoding SEQ ID NO:Y or a fragment or variant thereof (e.g., the polypeptide delinated in columns fourteen and fifteen of Table 1A); a nucleic acid sequence contained in SEQ ID NO:X (as described in column 5 of Table 1A and/or column 3 of Table 1B) or the complement thereof; a cDNA sequence contained in Clone ID: (as described in column 2 of Table 1A and/or Table 1B and contained within a library deposited with the ATCC); a nucleotide sequence encoding the polypeptide encoded by a nucleotide sequence in SEQ ID NO:B as defined in column 6 (EXON From-To) of Table 1C or a fragment or variant thereof; or a nucleotide coding sequence in SEQ ID NO:B as defined in column 6 of Table 1C or the complement thereof. For example, the polynucleotide can contain the nucleotide sequence of the full length cDNA sequence, including the 5' and 3' untranslated sequences, the coding region, as well as fragments, epitopes, domains, and variants of the nucleic acid sequence. Moreover, as used herein, a "polypeptide" refers to a molecule having an amino acid sequence encoded by a polynucleotide of the invention as broadly defined (obviously excluding poly-Phenylalanine or poly-Lysine peptide sequences which result from translation of a polyA tail of a sequence corresponding to a cDNA).

In the present invention, "SEQ ID NO:X" was often generated by overlapping sequences contained in multiple clones (contig analysis). A representative clone containing all or most of the sequence for SEQ ID NO:X is deposited at Human Genome Sciences, Inc. (HGS) in a catalogued and archived library. As shown, for example, in column 2 of Table 1B, each clone is identified by a cDNA Clone ID (identifier generally referred to herein as Clone ID:). Each Clone ID is unique to an individual clone and the Clone ID is all the information needed to retrieve a given clone from the HGS library. Table 7 provides a list of the deposited cDNA libraries. One can use the Clone ID: to determine the library source by reference to Tables 6 and 7. Table 7 lists the deposited cDNA libraries by name and links each library to an ATCC Deposit. Library names contain four characters, for example, "HTWE." The name of a cDNA clone (Clone ID) isolated from that library begins with the same four characters, for example "HTWEP07". As mentioned below, Table 1A and/or Table 1B correlates the Clone ID names with SEQ ID NO:X. Thus, starting with an SEQ ID NO:X, one can use Tables 1A, 1B, 6, 7, and 9 to determine the corresponding Clone ID, which library it came from and which ATCC deposit the library is contained in. Furthermore,

it is possible to retrieve a given cDNA clone from the source library by techniques known in the art and described elsewhere herein. The ATCC is located at 10801 University Boulevard, Manassas, Virginia 20110-2209, USA. The ATCC deposits were made pursuant to the terms of the Budapest Treaty on the international recognition of the deposit of microorganisms for the purposes of patent procedure.

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In specific embodiments, the polynucleotides of the invention are at least 15, at least 30, at least 50, at least 100, at least 125, at least 500, or at least 1000 continuous nucleotides but are less than or equal to 300 kb, 200 kb, 100 kb, 50 kb, 15 kb, 10 kb, 7.5kb, 5 kb, 2.5 kb, 2.0 kb, or 1 kb, in length. In a further embodiment, polynucleotides of the invention comprise a portion of the coding sequences, as disclosed herein, but do not comprise all or a portion of any intron. In another embodiment, the polynucleotides comprising coding sequences do not contain coding sequences of a genomic flanking gene (i.e., 5' or 3' to the gene of interest in the genome). In other embodiments, the polynucleotides of the invention do not contain the coding sequence of more than 1000, 500, 250, 100, 50, 25, 20, 15, 10, 5, 4, 3, 2, or 1 genomic flanking gene(s).

A "polynucleotide" of the present invention also includes those polynucleotides capable of hybridizing, under stringent hybridization conditions, to sequences contained in SEQ ID NO:X, or the complement thereof (e.g., the complement of any one, two, three, four, or more of the polynucleotide fragments described herein), the polynucleotide sequence delineated in columns 7 and 8 of Table 1A or the complement thereof, the polynucleotide sequence delineated in columns 8 and 9 of Table 2 or the complement thereof, and/or cDNA sequences contained in Clone ID: (e.g., the complement of any one, two, three, four, or more of the polynucleotide fragments, or the cDNA clone within the pool of cDNA clones deposited with the ATCC, described herein), and/or the polynucleotide sequence delineated in column 6 of Table 1C or the complement thereof. "Stringent hybridization conditions" refers to an overnight incubation at 42 degree C in a solution comprising 50% formamide, 5x SSC (750 mM NaCl, 75 mM trisodium citrate), 50 mM sodium phosphate (pH 7.6), 5x Denhardt's solution, 10% dextran sulfate, and 20 μg/ml denatured, sheared salmon sperm DNA, followed by washing the filters in 0.1x SSC at about 65 degree C.

Also contemplated are nucleic acid molecules that hybridize to the polynucleotides of the present invention at lower stringency hybridization conditions. Changes in the stringency of hybridization and signal detection are primarily accomplished through the manipulation of formamide concentration (lower percentages of formamide result in lowered stringency); salt conditions, or temperature. For example, lower stringency conditions include an overnight incubation at 37 degree C in a solution comprising 6X SSPE (20X SSPE = 3M NaCl; 0.2M NaH₂PO₄; 0.02M EDTA, pH 7.4), 0.5% SDS, 30% formamide, 100 ug/ml salmon sperm blocking DNA; followed by washes at 50 degree C with 1XSSPE, 0.1% SDS. In addition, to achieve even lower stringency, washes performed following stringent hybridization can be done at higher salt concentrations (e.g. 5X SSC).

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Note that variations in the above conditions may be accomplished through the inclusion and/or substitution of alternate blocking reagents used to suppress background in hybridization experiments. Typical blocking reagents include Denhardt's reagent, BLOTTO, heparin, denatured salmon sperm DNA, and commercially available proprietary formulations. The inclusion of specific blocking reagents may require modification of the hybridization conditions described above, due to problems with compatibility.

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Of course, a polynucleotide which hybridizes only to polyA+ sequences (such as any 3' terminal polyA+ tract of a cDNA shown in the sequence listing), or to a complementary stretch of T (or U) residues, would not be included in the definition of "polynucleotide," since such a polynucleotide would hybridize to any nucleic acid molecule containing a poly (A) stretch or the complement thereof (e.g., practically any double-stranded cDNA clone generated using oligo dT as a primer).

The polynucleotide of the present invention can be composed of any polyribonucleotide or polydeoxribonucleotide, which may be unmodified RNA or DNA or modified RNA or DNA. For example, polynucleotides can be composed of single- and double-stranded DNA, DNA that is a mixture of single- and double-stranded regions, single- and double-stranded RNA, and RNA that is mixture of single- and double-stranded regions, hybrid molecules comprising DNA and RNA that may be single-stranded or, more typically, double-stranded or a mixture of single- and double-stranded regions. In addition, the polynucleotide can be composed of triple-stranded regions comprising RNA or DNA or both RNA and DNA. A polynucleotide may also contain one or more modified bases or DNA or RNA backbones modified for stability or for other reasons. "Modified" bases include, for example, tritylated bases and unusual bases such as inosine. A variety of modifications can be made to DNA and RNA; thus, "polynucleotide" embraces chemically, enzymatically, or metabolically modified forms.

In specific embodiments, the polynucleotides of the invention are at least 15, at least 30, at least 50, at least 100, at least 125, at least 500, or at least 1000 continuous nucleotides but are less than or equal to 300 kb, 200 kb, 100 kb, 50 kb, 15 kb, 10 kb, 7.5kb, 5 kb, 2.5 kb, 2.0 kb, or 1 kb, in length. In a further embodiment, polynucleotides of the invention comprise a portion of the coding sequences, as disclosed herein, but do not comprise all or a portion of any intron. In another embodiment, the polynucleotides comprising coding sequences do not contain coding sequences of a genomic flanking gene (i.e., 5' or 3' to the gene of interest in the genome). In other embodiments, the polynucleotides of the invention do not contain the coding sequence of more than 1000, 500, 250, 100, 50, 25, 20, 15, 10, 5, 4, 3, 2, or 1 genomic flanking gene(s).

"SEQ ID NO:X" refers to a polynucleotide sequence described in column 5 of Table 1A, while "SEQ ID NO:Y" refers to a polypeptide sequence described in column 10 of Table 1A. SEQ ID NO:X is identified by an integer specified in column 6 of Table 1A. The polypeptide sequence SEQ ID NO:Y is a translated open reading frame (ORF) encoded by polynucleotide SEQ ID

NO:X. The polynucleotide sequences are shown in the sequence listing immediately followed by all of the polypeptide sequences. Thus, a polypeptide sequence corresponding to polynucleotide sequence SEQ ID NO:2 is the first polypeptide sequence shown in the sequence listing. The second polypeptide sequence corresponds to the polynucleotide sequence shown as SEQ ID NO:3, and so on.

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The polypeptide of the present invention can be composed of amino acids joined to each other by peptide bonds or modified peptide bonds, i.e., peptide isosteres, and may contain amino acids other than the 20 gene-encoded amino acids. The polypeptides may be modified by either natural processes, such as posttranslational processing, or by chemical modification techniques which are well known in the art. Such modifications are well described in basic texts and in more detailed monographs, as well as in a voluminous research literature. Modifications can occur anywhere in a polypeptide, including the peptide backbone, the amino acid side-chains and the amino or carboxyl termini. It will be appreciated that the same type of modification may be present in the same or varying degrees at several sites in a given polypeptide. Also, a given polypeptide may contain many types of modifications. Polypeptides may be branched, for example, as a result of ubiquitination, and they may be cyclic, with or without branching. Cyclic, branched, and branched cyclic polypeptides may result from posttranslation natural processes or may be made by synthetic methods. Modifications include acetylation, acylation, ADPribosylation, amidation, covalent attachment of flavin, covalent attachment of a heme moiety, covalent attachment of a nucleotide or nucleotide derivative, covalent attachment of a lipid or lipid derivative, covalent attachment of phosphotidylinositol, cross-linking, cyclization, disulfide bond formation, demethylation, formation of covalent cross-links, formation of cysteine, formation of pyroglutamate, formylation, gamma-carboxylation, glycosylation, GPI anchor formation, hydroxylation, iodination, methylation, myristoylation, oxidation, pegylation, proteolytic processing, phosphorylation, prenylation, racemization, selenoylation, sulfation, transfer-RNA mediated addition of amino acids to proteins such as arginylation, and ubiquitination. (See, for instance, PROTEINS - STRUCTURE AND MOLECULAR PROPERTIES, 2nd Ed., T. E. Creighton, W. H. Freeman and Company, New York (1993); POSTTRANSLATIONAL COVALENT MODIFICATION OF PROTEINS, B. C. Johnson, Ed., Academic Press, New York, pgs. 1-12 (1983); Seifter et al., Meth. Enzymol. 182:626-646 (1990); Rattan et al., Ann. N.Y. Acad. Sci. 663:48-62 (1992)).

"SEQ ID NO:X" refers to a polynucleotide sequence described, for example, in Tables 1A, Table 1B, or Table 2, while "SEQ ID NO:Y" refers to a polypeptide sequence described in column 11 of Table 1A and or column 6 of Table 1B.1,. SEQ ID NO:X is identified by an integer specified in column 4 of Table 1B. The polypeptide sequence SEQ ID NO:Y is a translated open reading frame (ORF) encoded by polynucleotide SEQ ID NO:X. "Clone ID:" refers to a cDNA clone described in column 2 of Table 1A and/or 1B.

"A polypeptide having functional activity" refers to a polypeptide capable of displaying one or more known functional activities associated with a full-length (complete) protein. Such functional activities include, but are not limited to, biological activity (e.g. activity useful in treating, preventing and/or ameliorating hematopoietic and hematologic diseases and disorders), antigenicity (ability to bind [or compete with a polypeptide for binding] to an anti-polypeptide antibody), immunogenicity (ability to generate antibody which binds to a specific polypeptide of the invention), ability to form multimers with polypeptides of the invention, and ability to bind to a receptor or ligand for a polypeptide.

The polypeptides of the invention can be assayed for functional activity (e.g. biological activity) using or routinely modifying assays known in the art, as well as assays described herein. Specifically, one of skill in the art may routinely assay secreted polypeptides (including fragments and variants) of the invention for activity using assays as described in the examples section below.

"A polypeptide having biological activity" refers to a polypeptide exhibiting activity similar to, but not necessarily identical to, an activity of a polypeptide of the present invention, including mature forms, as measured in a particular biological assay, with or without dose dependency. In the case where dose dependency does exist, it need not be identical to that of the polypeptide, but rather substantially similar to the dose-dependence in a given activity as compared to the polypeptide of the present invention (i.e., the candidate polypeptide will exhibit greater activity or not more than about 25-fold less and, preferably, not more than about tenfold less activity, and most preferably, not more than about three-fold less activity relative to the polypeptide of the present invention).

TABLES

25 Table 1A

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Table 1A summarizes information concerning certain polypnucleotides and polypeptides of the invention. The first column provides the gene number in the application for each clone identifier. The second column provides a unique clone identifier, "Clone ID:", for a cDNA clone related to each contig sequence disclosed in Table 1A. Third column, the cDNA Clones identified in the second column were deposited as indicated in the third column (i.e. by ATCC Deposit No:Z and deposit date). Some of the deposits contain multiple different clones corresponding to the same gene. In the fourth column, "Vector" refers to the type of vector contained in the corresponding cDNA Clone identified in the second column. In the fifth column, the nucleotide sequence identified as "NT SEQ ID NO:X" was assembled from partially homologous ("overlapping") sequences obtained from the corresponding cDNA clone identified in the second column and, in some cases, from additional related cDNA clones. The overlapping sequences were assembled into a single contiguous sequence of high redundancy (usually three to five

overlapping sequences at each nucleotide position), resulting in a final sequence identified as SEQ ID NO:X. In the sixth column, "Total NT Seq." refers to the total number of nucleotides in the contig sequence identified as SEQ ID NO:X." The deposited clone may contain all or most of these sequences, reflected by the nucleotide position indicated as "5' NT of Clone Seq." (seventh column) and the "3' NT of Clone Seq." (eighth column) of SEQ ID NO:X. In the ninth column, the nucleotide position of SEQ ID NO:X of the putative start codon (methionine) is identified as "5' NT of Start Codon." Similarly, in column ten, the nucleotide position of SEQ ID NO:X of the predicted signal sequence is identified as "5' NT of First AA of Signal Pep." In the eleventh column, the translated amino acid sequence, beginning with the methionine, is identified as "AA SEQ ID NO:Y," although other reading frames can also be routinely translated using known molecular biology techniques. The polypeptides produced by these alternative open reading frames are specifically contemplated by the present invention.

In the twelfth and thirteenth columns of Table 1A, the first and last amino acid position of SEQ ID NO:Y of the predicted signal peptide is identified as "First AA of Sig Pep" and "Last AA of Sig Pep." In the fourteenth column, the predicted first amino acid position of SEQ ID NO:Y of the secreted portion is identified as "Predicted First AA of Secreted Portion". The amino acid position of SEQ ID NO:Y of the last amino acid encoded by the open reading frame is identified in the fifteenth column as "Last AA of ORF".

SEQ ID NO:X (where X may be any of the polynucleotide sequences disclosed in the sequence listing) and the translated SEQ ID NO:Y (where Y may be any of the polypeptide sequences disclosed in the sequence listing) are sufficiently accurate and otherwise suitable for a variety of uses well known in the art and described further below. For instance, SEQ ID NO:X is useful for designing nucleic acid hybridization probes that will detect nucleic acid sequences contained in SEQ ID NO:X or the cDNA contained in the deposited clone. These probes will also hybridize to nucleic acid molecules in biological samples, thereby enabling a variety of forensic and diagnostic methods of the invention. Similarly, polypeptides identified from SEQ ID NO:Y may be used, for example, to generate antibodies which bind specifically to proteins containing the polypeptides and the secreted proteins encoded by the cDNA clones identified in Table 1A and/or elsewhere herein

Nevertheless, DNA sequences generated by sequencing reactions can contain sequencing errors. The errors exist as misidentified nucleotides, or as insertions or deletions of nucleotides in the generated DNA sequence. The erroneously inserted or deleted nucleotides cause frame shifts in the reading frames of the predicted amino acid sequence. In these cases, the predicted amino acid sequence diverges from the actual amino acid sequence, even though the generated DNA sequence may be greater than 99.9% identical to the actual DNA sequence (for example, one base insertion or deletion in an open reading frame of over 1000 bases).

Accordingly, for those applications requiring precision in the nucleotide sequence or the amino acid sequence, the present invention provides not only the generated nucleotide sequence identified as SEQ ID NO:X, and the predicted translated amino acid sequence identified as SEQ ID NO:Y, but also a sample of plasmid DNA containing a human cDNA of the invention deposited with the ATCC, as set forth in Table 1A. The nucleotide sequence of each deposited plasmid can readily be determined by sequencing the deposited plasmid in accordance with known methods

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The predicted amino acid sequence can then be verified from such deposits. Moreover, the amino acid sequence of the protein encoded by a particular plasmid can also be directly determined by peptide sequencing or by expressing the protein in a suitable host cell containing the deposited human cDNA, collecting the protein, and determining its sequence.

Also provided in Table 1A is the name of the vector which contains the cDNA plasmid. Each vector is routinely used in the art. The following additional information is provided for convenience.

Vectors Lambda Zap (U.S. Patent Nos. 5,128,256 and 5,286,636), Uni-Zap XR (U.S. Patent Nos. 5,128, 256 and 5,286,636), Zap Express (U.S. Patent Nos. 5,128,256 and 5,286,636), pBluescript (pBS) (Short, J. M. et al., Nucleic Acids Res. 16:7583-7600 (1988); Alting-Mees, M. A. and Short, J. M., Nucleic Acids Res. 17:9494 (1989)) and pBK (Alting-Mees, M. A. et al., Strategies 5:58-61 (1992)) are commercially available from Stratagene Cloning Systems, Inc., 11011 N. Torrey Pines Road, La Jolla, CA, 92037. pBS contains an ampicillin resistance gene and pBK contains a neomycin resistance gene. Phagemid pBS may be excised from the Lambda Zap and Uni-Zap XR vectors, and phagemid pBK may be excised from the Zap Express vector. Both phagemids may be transformed into E. coli strain XL-1 Blue, also available from Stratagene

Vectors pSport1, pCMVSport 1.0, pCMVSport 2.0 and pCMVSport 3.0, were obtained from Life Technologies, Inc., P. O. Box 6009, Gaithersburg, MD 20897. All Sport vectors contain an ampicillin resistance gene and may be transformed into E. coli strain DH10B, also available from Life Technologies. See, for instance, Gruber, C. E., et al., Focus 15:59 (1993). Vector lafmid BA (Bento Soares, Columbia University, New York, NY) contains an ampicillin resistance gene and can be transformed into E. coli strain XL-1 Blue. Vector pCR®2.1, which is available from Invitrogen, 1600 Faraday Avenue, Carlsbad, CA 92008, contains an ampicillin resistance gene and may be transformed into E. coli strain DH10B, available from Life Technologies. See, for instance, Clark, J. M., Nuc. Acids Res. 16:9677-9686 (1988) and Mead, D. et al., Bio/Technology 9: (1991).

The present invention also relates to the genes corresponding to SEQ ID NO:X, SEQ ID NO:Y, and/or a deposited cDNA (cDNA Clone ID). The corresponding gene can be isolated in accordance with known methods using the sequence information disclosed herein. Such methods

include, but are not limited to, preparing probes or primers from the disclosed sequence and identifying or amplifying the corresponding gene from appropriate sources of genomic material.

Also provided in the present invention are allelic variants, orthologs, and/or species homologs. Procedures known in the art can be used to obtain full-length genes, allelic variants, splice variants, full-length coding portions, orthologs, and/or species homologs of genes corresponding to SEQ ID NO:X and SEQ ID NO:Y using information from the sequences disclosed herein or the clones deposited with the ATCC. For example, allelic variants and/or species homologs may be isolated and identified by making suitable probes or primers from the sequences provided herein and screening a suitable nucleic acid source for allelic variants and/or the desired homologue.

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The present invention provides a polynucleotide comprising, or alternatively consisting of, the nucleic acid sequence of SEQ ID NO:X and/or a cDNA contained in ATCC Deposit No.Z. The present invention also provides a polypeptide comprising, or alternatively, consisting of, the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X, and/or a polypeptide encoded by a cDNA contained in ATCC deposit No.Z. Polynucleotides encoding a polypeptide comprising, or alternatively consisting of the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X and/or a polypeptide encoded by the cDNA contained in ATCC Deposit No.Z, are also encompassed by the invention. The present invention further encompasses a polynucleotide comprising, or alternatively consisting of the complement of the nucleic acid sequence of SEQ ID NO:X, and/or the complement of the coding strand of the cDNA contained in ATCC Deposit No.Z.

FABLE 1A

		-		I										
				Z		5. NT 3. NT	3. NT		5' NT of First	AA	First	Last		
		ATCC		SEO		Jo	of	5° NT	AA of			AA of	First AA	Last
		Deposit No:Z		Α	Total	Clone	Clone	Clone of Start Signal	Signal		Sig	Sig	ot	AA of
ene	cDNA	and Date		ö	Ņ	Seq.	Seq.	Codon	Pep	NO:Y	Pep	Pep	Secreted	ORF
So.	Clone ID		Vector	×	Seq.								Portion	
_	H2CBU83	209889	pBluescript	11	2703	1	2703	157	157	979	1	30	31	207
		05/22/98	SK-											
_	H2CBU83	209889	pBluescript	402	2709	1	2709	157	157	1017	_	30	31	51
		05/22/98	SK-											
2	H2MAC30	209299	pBluescript	12	459	1	654	157	157	627		78	29	72
		09/25/97	SK-											
3	H6EAB28	209511	Uni-ZAP XR	13	1939	1	1939	115	115	628	-	31	32	8
		12/03/97												
6	H6EAB28	209511	Uni-ZAP XR	403	1547	1	1547	116	911	1018	-	20	21	92
		12/03/97												
4	H6EDC19	209324	Uni-ZAP XR	14	092	324	992	389	389	629	-	25	5 6	114
		10/02/97												
2	H6EDF66	209299	Uni-ZAP XR	15	540	T	540	146	146	630	1	27	78	131
		09/25/97												
9	H6EDX46	209626	Uni-ZAP XR	91	888	1	888	229	. 229	631		70	21	182
		02/12/98				į								
9	H6EDX46	209626	Uni-ZAP XR	404	718	-	718	128	128	1019		20	21	84
		02/12/98												
7	HACBD91	209626	Uni-ZAP XR	17	1445	1	1445	117	117	632	1	42	43	49
·		02/12/98												
∞	HACCI17	203071	Uni-ZAP XR	81	1722	336	1714	461	461	633	-	24	25	218
		07/27/98												

	Gene No.	∞	9	01	=	12	17	13	13	4	15
	cDNA Clone ID	HACCI17	HAGAI85	HAGAQ26	HAGDI35	HAGDS35	HAGDS35	HAGFY16	HAGFY16	HAIBO71	HAIBP89
ATCC	Deposit No:Z and Date	203071 07/27/98	97922 03/07/97 209070 05/22/97	209368 10/16/97	209852 05/07/98	209299 09/25/97	209299 09/25/97	97923 03/07/97 209071 05/22/97	97923 03/07/97 209071 05/22/97	209145 07/17/97	209877 05/18/98
	Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
NT SEQ	АÄ×	405	19	20	21	22	406	23	407	24	25
	Total NT Seq.	1380	1752	1333	1357	751	813	1963	1830	752	2243
S' NT 3' NT of	Clone Seq.	12	52	157	-		1	209	87	172	173
	Clone Seq.	1380	1752	1333	1338	751	813	1922	1786	752	2243
S' NT	Clone of Start Signal Seq. Codon Pep	135	166	251	318	45	25	251	128	325	311
5' NT of First AA of	Signal Pep	135	166	251	318	45	25	251	128	325	311
AA SEQ	ID NO:Y	1020	634	635	636	637	1021	638	1022	629	640
First AA of	Sig Pep	1	-	1	1	1	1	1		1	-
First Last AA of AA of	Sig Pep	24	23	20	25	23	23	28	26	28	27
First AA	of Secreted Portion	25	24	21	56	24	24	53	27	29	28
Last	AA of ORF	72	30	62	93	122	118	861	45	99	317

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Last AA of	ORF	18	446	563	169	53	607	161	178	80	421	47	79	242
First AA	Secreted Portion	2	19	16	16	20	23	31	31		35	24	32	61
Last AA of Sig	Pep	1	18	15	15	19	22	30	30		34	23	31	18
First AA of Sig	Pep	-		1	1	1	1	1	1	1	1	1	1	-
AA SEQ U	NO:Y	1023	641	642	1024	643	644	645	1025	1026	646	1027	647	648
5' NT of First AA of Signal	Pep	1	128	109	120	262	49	981	115	323	1495	526	279	86
3' NT of 5' NT Clone of Start	Codon		128	109	120	262	49	136	115		1495	226	279	86
3' NT of Clone	Seq.	1025	1483	2849	2288	755	2085	2534	824	3941	4129	3758	785	1674
5' NT of Clone	Seq.	1	68	1	-		10	1	-	1947	1	1	1	47
Total	NT Seq.	1025	1624	2849	2288	755	5089	2534	824	3941	4129	3758	785	1674
NT SEQ	ÿ×	408	26	27	409	28	29	30	410	411	31	412	32	33
	Vector	Uni-ZAP XR	Uni-ZAP XR	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0
ATCC	and Date	209877 05/18/98	209009 04/28/97	PTA-322 07/09/99	PTA-322 07/09/99	209626 02/12/98	209603 01/29/98	PTA-849 10/13/99	PTA-849 10/13/99	PTA-849 10/13/99	203364 10/19/98	203364 10/19/98	209641 02/25/98	209965 06/11/98
	cDNA Clone ID	HAIBP89	HAICP19	HAJAN23	HAJAN23	HAJBR69	HAJBZ75	HAMFC93	HAMFC93	HAMFC93	HAMFE15	HAMFE15	HAMFK58	HAMGR28
	Gene No.	15	16	17	17	18	19	20	20	20	21	21	22	23

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Last AA of ORF	203	52	86	392	434	189	123	264	91	140	248	189	49
First AA of Secreted Portion	19	2	24	30	27	23	2	23	23	26	22	22	17
Last AA of Sig Pep	18	1	23	29	26	22	1	22	22	25	21	21	16
First AA of Sig Pep	1	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ ID NO:Y	1028	649	1029	1030	1031	029	1032	651	1033	652	653	1034	654
5' NT of First AA of Signal Pep	40	520	125	70	78	251	448	59	54	385	26	66	252
5' NT of Start Codon	40	520	125	70	78	251	448	59	54	385	97	66	252
3' NT of Clone Seq.	1534	2398	2454	1775	1379	2005	2664	1472 _.	1501	1153	1959	1306	812
of of Clone Seq.	1	348	1	-	-	1	1	1	14	1	1	13	1
Total NT Seq.	1534	2657	2454	3//1	1379	2005	2664	1472	1508	1153	1959	1306	812
× Signal X	413	34	414	415	416	35	417	36	418	37	38	419	39
Vector	pCMVSport 3.0	pSport1	pSport1	pSport1	pSport1	Uni-ZAP XR							
ATCC Deposit No:Z and Date	209965 06/11/98	PTA-1543 03/21/00	PTA-1543 03/21/00	PTA-1543 03/21/00	PTA-1543 03/21/00	209878 05/18/98	209878 05/18/98	209683 03/20/98	209683 03/20/98	203570 01/11/99	209651 03/04/98	209651 03/04/98	209626 02/12/98
cDNA Clone ID	HAMGR28	HANGG89	HANGG89	HANGG89	HANGG89	HAPOM49	HAPOM49	HAPPW30	HAPPW30	HAPUC89	HATAC53	HATAC53	HATBR65
Gene No.	23	24	24	24	24	25	25	56	26	27	28	28	29

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Last AA of ORF	385	53	49	23	110	258	65	202	196	08	80	240	09
First AA of Secreted Portion	31	22	19	11	16	2	20	38	38	21	21	39	39
Last AA of Sig Pep	30	21	18	10	15	1	19	37	37	20	20	38	38
First AA of Sig Pep	1	1	1	1	1	1	1	1	1	1	-	1	1
AA SEQ ID NO:Y	655	959	657	1035	658	629	099	661	1036	662	1037	663	1038
5' NT of First AA of Signal Pep	143	241	253	575	09	9	390	88	68	26	760	124	62
3' NT of 5' NT Clone of Start Seq. Codon	143	241	253		09	9	390	88	68	26	760	124	62
3' NT of Clone Seq.	1355	863	988	1076	1252	953	821	1368	729	402	1180	981	933
5' NT of Clone Seq.	1	136	1	1	1	1	330	-	1	1	741	1	1
Total NT Seq.	1355	1675	910	1076	1280	953	821	1368	729	402	1180	981	943
SEQ SEQ SO:	40	41	42	420	43	44	45	46	421	47	422	84 80	423
Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pSportl	pSport1	pSport1	pSport1	pSport1	pSport1	pSport1	Uni-ZAP XR	Uni-ZAP XR
ATCC Deposit No:Z and Date	203858 03/18/99	209407 10/23/97	209626 02/12/98	209626 02/12/98	209603 01/29/98	PTA-1543 03/21/00	209683 03/20/98	PTA-2075 06/09/00	PTA-2075 06/09/00	PTA-2075 06/09/00	PTA-2075 06/09/00	209878 05/18/98	209878 05/18/98
cDNA Clone ID	HATDF29	HATEE46	HAUAI83	HAUAI83	HBAF133	HBAFV19	HBAMB15	HBCPB32	НВСРВ32	нвсог32	нвсог32	HBGBA69	HBGBA69
Gene No.	30	31	32	32	33	34	35	36	36	37	37	38	38

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Last AA of ORF	33	146	130	174	173	210	199	111	84	245	66	245
First AA of Secreted Portion	19	22	22	31	30	30	21	21	61	23	23	23
Last AA of Sig Pep	18	21	21	30	29	29	20	20	18	22	22	22
First AA of Sig Pep	1	1	1	1	1	-		1	1	1	П	1
AA SEQ ID NO:Y	664	999	1039	999	1040	1041	<i>1</i> 99	1042	899	699	1043	1044
5' NT of First AA of Signal Pep	75	86	93	57	71	100	157	137	133	99	99	2
3' NT of 5' NT Clone of Start Seq. Codon	75	86	93	57	71	001	157	137	133	99	99	64
3' NT of Clone Seq.	1038	537	526	843	1566	1067	2008	571	1160	1061	1021	1023
5' NT 3' NT of of Clone Clone Seq.	1	-	1	1	-	1	1	1	1	1	1	1
Total NT Seq.	1038	537	526	843	1566	1067	2008	571	1160	1061	1021	1086
F S B S ×	49	20	424	51	425	426	52	427	53	χ	428	429
Vector	Uni-ZAP XR	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC Deposit No:Z and Date	209224 08/28/97	209683 03/20/98	209683 03/20/98	PTA-885 10/28/99	PTA-885 10/28/99	PTA-885 10/28/99	209300 09/25/97	209300 09/25/97	209346 10/09/97	PTA-622 09/02/99	PTA-622 09/02/99	PTA-622 09/02/99
cDNA Clone ID	HBIAE26	HBIMB51	HBIMB51	HBINS58	HBINS58	HBINS58	HBJID05	HBJID05	HBJJU28	HBJNC59	HBJNC59	HBJNC59
Gene No.	39	40	9	41	14	41	42	42	43	4	44	4

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Last AA of ORF	83	19	57	83	56	319	336	105	272	09	224	68
First AA of Secreted Portion	30	38	32	31	19	20	20	20	21	24	25	36
Last AA of Sig Pep	29	37	31	30	18	19	19	19	20	23	24	35
First AA of Sig Pep	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ ID NO:Y	0.29	671	672	673	674	919	9/9	1045	<i>LL</i> 9	8/9	619	089
5' NT of First AA of Signal Pep	156	11	995	137	891	991	165	165	113	134	188	12
5' NT of Start Codon	156	11	260	137	168	166	165	165	113	134	188	12
3' NT of Clone Seq.	920	601	2210	1554	427	1256	2084	2078	1765	1016	1430	2494
S' NT of Clone Seq.	1	1	376	1	ı	19	-			-		
Total NT Seq.	920	601	5229	1554	427	1276	2084	2078	1765	1016	1430	2494
SEQ SEQ	55	26	57	28	59	9	19	430	62	63	49	92
Vector	pBluescript	Uni-ZAP XR	ZAP Express	Uni-ZAP XR								
ATCC Deposit No:Z and Date	97978 03/27/97 209075 05/22/97	209242 09/12/97	203858 03/18/99	209626 02/12/98	209300 09/25/97	209626 02/12/98	209878 05/18/98	209878 05/18/98	209580 01/14/98	209626 02/12/98	209965 06/11/98	PTA-2069 06/09/00
cDNA Clone ID	HBMCI50	HBNAW17	HBXFL29	HCACU58	HCDAF84	HCE2F54	HCE3G69	HCE3G69	HCESF43	HCEEA88	HCEFB69	HCEFB80
Gene No.	45	46	47	48	49	20	51	51	52	53	54	55

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Last	AA of ORF	68	31	106	27	54	51	74	88	127	127	6	215	91
臣	of Secreted Portion	36	19	24	20	2	19	31	20	25	48	6	27	27
Last AA of	Sig Pep	35	18	23	19	1	18	30	61	24	47	8	26	26
First AA of	Sig Pep	1	-		1	1	1	1	1	1	1	-	-	1
AA SEQ	NO:Y	1046	681	682	1047	1048	683	684	685	989	1049	1050	289	1051
S' NT of First AA of	Signal Pep	5	243	117	500	156	166	438	148	21	124	603	107	161
	of Start Codon	5	243	117	200		166	438	148	21	124		107	161
3' NT of	Clone Seq.	2451	1630	296	730	550	885	780	1262	1343	845	738	1089	1145
5' NT of	Clone Seq.	1	1	1	247	1	13	1	1	1	1	-	_	62
	Total NT Seq.	2494	1630	<i>L</i> 96	730	550	885	790	1262	1343	845	738	1089	1145
NT SEQ	ВÄ×	431	99	<i>L</i> 9	432	433	89	69	70	71	434	435	72	436
	Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pCMVSport 2.0	Uni-ZAP XR	Lambda ZAP II	Lambda ZAP II	Lambda ZAP II	pBluescript	pBluescript
ATCC	Deposit No:Z and Date	PTA-2069 06/09/00	209090 06/05/97	PTA-842 10/13/99	PTA-842 10/13/99	PTA-842 10/13/99	209300	209627 02/12/98	209877 05/18/98	PTA-855 10/18/99	PTA-855 10/18/99	PTA-855 10/18/99	209580	209580 01/14/98
	cDNA Clone ID	HCEFB80	HCEGR33	HCEWE17	HCEWE17	HCEWE17	HCEWE20	HCGMD59	HCMSQ56	HCNDR47	HCNDR47	HCNDR47	HCNSM70	HCNSM70
	Gene No.	55	26	57	57	57	58	29	99	61	61	61	62	62

Last AA of ORF	158	158	106	47	100	343	244	244	40	65	274	108	413
First AA of A Secreted	27	27	2	28	18	28	28	28	19	22	22	25	34
Last AA of Sig Pep	26	26	-	27	17	27	27	27	18	21	21	24	33
First AA of Sig Pep	-	1	1	-	1	1	1	1	1	1	1	1	1
AA SEQ ID NO:Y	889	1052	1053	689	069	691	1054	1055	692	693	694	695	969
5' NT of First AA of Signal Pep	36	40	1	557	19	148	247	155	37	138	118	568	199
5' NT of Start Codon	36	40		557	19	148	247	155	37	138	118	568	199
3' NT of Clone Seq.	1254	869	909	736	320	1283	086	888	710	1540	2661	1421	2184
S' NT of Clone Seq.	-	15	339	331	1	-	1	-	1	1	-	235	1
Total NT Sea	1254	698	692	875	320	1283	086	888	710	1540	3061	1421	2184
SEQ X	73	437	438	74	75	9/	439	440	11	78	62	08	81
Vector	pSport1	pSport	pSportl	ZAP Express	ZAP Express	ZAP Express	ZAP Express	ZAP Express	ZAP Express	ZAP Express	pBluescript SK-	pCMVSport 2.0	pCMVSport 2.0
ATCC Deposit No:Z and Date	PTA-2076 06/09/00	PTA-2076 06/09/00	PTA-2076 06/09/00	209324 10/02/97	209852 05/07/98	PTA-883 10/28/99	PTA-883 10/28/99	PTA-883 10/28/99	209324 10/02/97	209626 02/12/98	209563	209215	203331 10/08/98
cDNA Ugue III	HCOOS80	HCOOS80	HCOOS80	HCUIM65	HCWDS72	HCWEB58	HCWEB58	HCWEB58	HCWKC15	HCWLD74	HCYBG92	нрнев60	HDHMA45
Gene	63	63	63	2	65	99	99	99	<i>L</i> 9	89	69	20	71

Last	AA of ORF	413	941	941	267	157	118	23	578	264	152	316	302	196
First AA	of Secreted Portion	34	33	33	59	18	7	21	22	22	20	29	19	19
Last AA of	Sig Pep	33	32	32	58	17	6	20	21	21	19	28	18	18
First AA of	Sig Pep	1	1	1	1	1	1	1	1	1	-	-	1	-
AA SEQ	NO:Y	1056	697	1057	698	1058	1059	699	700	1060	701	1061	1062	702
5' NT of First AA of	Signal Pep	204	259	69	35	260	605	182	76	76	173	681	218	175
S' NT	of Start Signa Codon Pep	204	259	69	35	260		182	9/	76	173	139	218	175
3' NT of	Clone Seq.	2190	3447	4909	3037	2921	1259	191	1932	1931	3436	1517	2751	1256
5' NT of	Clone Seq.	1	197	1	115	-	358	92	45	45	1	1	1976	1
	Total NT Seq.	2190	3447	4906	3037	2921	1259	191	1932	1931	3436	1517	2751	1256
NT SEQ	ВÄ×	441	82	442	83	443	4 4 4 4 4 4 4 4 4 4 4 4 4 4 4 4 4 4 4	25	85	445	98	446	447	87
	Vector	pCMVSport 2.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0
ATCC	Deposit No:Z and Date	203331 10/08/98	PTA-163 06/01/99	PTA-163 06/01/99	PTA-1544 03/21/00	PTA-1544 03/21/00	PTA-1544 03/21/00	209125 06/19/97	209568 01/06/98	209568 01/06/98	PTA-622 09/02/99	PTA-622 09/02/99	PTA-622 09/02/99	209511 12/03/97
	cDNA Clone ID	HDHMA45	HDPBA28	HDPBA28	HDPCL63	HDPCL63	HDPCL63	HDPC025	HDPCY37	HDPCY37	HDPFB02	HDPFB02	HDPFB02	HDPFF39
	Gene No.	71	72	72	73	73	73	74	75	75	9/	76	9/	77

Last	AA of ORF	52	87	40	27	525	65	937	109	46	9	11	99	53
First AA	of Secreted Portion	31	29	31	24	09	21	38	21	∞		9	2	2
Last AA of	Sig Pep	30	28	30	23	59	20	37	20	L		5	I	1
First AA of	Sig Pep	1	1	1	1	1	1	1	I	1	1	1	1	-
AA SEQ	NO:Y	703	704	705	902	707	1063	708	1064	1065	1066	1067	1068	6901
5' NT of First AA of	Signal Pep	293	8	245	196	59	259	100	141	44	419	111	167	28
S' NT	of Start Codon	293	∞	245	196	59	259	100	141					
3° NT of	Clone Seq.	1057	2687	728	986	1633	1313	4893	468	181	612	1024	321	519
S' NT of	Clone Seq.	1	138	-	-	308	1	1	1	1	1	1	18	1
	Total NT Seq.	1057	2687	728	986	1635	1314	4893	468	181	612	1024	366	519
NT SEQ	ДÄХ	88	86	8	91	92	448	93	449	450	451	452	453	454
	Vector	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0
ATCC	Deposit No:Z and Date	209626 02/12/98	203027 06/26/98	209125 06/19/97	209852 05/07/98	209563 12/18/97	209563 12/18/97	PTA-848 10/13/99						
	cDNA Clone ID	HDPFP29	HDPGT01	HDPHI51	HDPJF37	HDPJM30	HDPJM30	HDPMM88						
	Gene No.	78	79	80	81	82	82	83	83	83	83	83	83	83

Last AA of	ORF	151	242	122	46	46	99	64	14	<i>L</i> 01	06	121	06	710
First AA of	Secreted Portion	35	30	19	33	27	19	18	8	7	20	20	20	21
Last AA of Sig	Рер	34	29	18	32	26	18	17	7	1	19	19	19	20
First AA of Sig	Pep	1	1	1	1	1	1	1	1	1	1	1	1	ı
AA SEQ B	NO:Y	709	710	711	712	1070	713	1071	1072	1073	714	1074	2/01	715
5' NT of First AA of Signal	Pep	118	252	159	127	117	123	116	1525	345	158	153	212	184
	Codon	118	252	159	127	117	123				158	153	212	184
	Seq.	1353	2504	1655	6297	2042	3408	308	1568	865	1663	1687	570	2343
	Seq.	1	1	1	1	1	1	1	1	1	1	1	1	1
Total	NT Seq.	1353	2504	1655	6297	2042	3408	308	1568	865	1663	1687	570	2343
SEQ	NO: X	94	95	96	6	455	86	456	457	458	66	459	460	100
	Vector	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0
ATCC Deposit No:Z	and Date	PTA-622 · 09/02/99	209745 04/07/98	209878 05/18/98	PTA-867 10/26/99	PTA-867 10/26/99	PTA-868 10/26/99	209745 04/07/98						
	cDNA Clone ID	HDPOE32	нороно6	HDPOJ08	HDPPN86	HDPPN86	HDPSB18	HDPSB18	HDPSB18	HDPSB18	HDPSH53	HDPSH53	HDPSH53	HDPSP01
	Gene No.	84	82	98	87	87	88	88	88	88	88	86	68	8

Last	ORF	308	48	55	467	455	66	802	214	43	86	86	22	25
First AA	Secreted Portion	21	19	42	19	29	29	20	20	20	38	38	10	17
First Last AA of AA of	Pep	20	18	41	18	28	28	61	19	61	37	37	6	16
First AA of Sio	Pep	-	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ	NO:Y	1076	716	1077	717	718	1078	719	1079	1080	720	1081	1082	1083
5' NT of First AA of Signal	Pep	227	2356	179	40	<i>L</i> 9	20	45	35	27	23	33	539	1190
3' NT of 5' NT	Codon	227	2356	179	40	<i>L</i> 9	20	45	35	27	23	33		
3. NT of	Seq.	1752	3091	536	1748	2339	397	2669	716	2716	992	2409	423	1471
S' NT of	Seq.	1	2304	1	-	1	-	1	1	76	1		-	105
T. 040	NT Seq.	1752	3091	536	1748	2339	397	2679	716	2716	992	2409	737	1471
SEQ	∃ ÿ×	461	101	462	102	103	463	<u>\$</u>	464	465	105	466	467	468
	Vector	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0
ATCC	Deposit No: 2 and Date	209745 04/07/98	209782 04/20/98	209782 04/20/98	203331 10/08/98	PTA-869 10/26/99	PTA-869 10/26/99	PTA-868 10/26/99						
	cDNA Clone ID	HDPSP01	HDPSP54	HDPSP54	HDPUW68	HDPVW11	HDPVW11	HDPWN93	HDPWN93	HDPWN93	HDPXY01	HDPXY01	HDPXY01	HDPXY01
	Gene No.	96	91	16	92	93	93	94	94	94	95	95	95	95

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Last	AA of ORF	331	333	365	365	219	220	809	95	108	73	29	29	80
First AA	of Secreted Portion	21	21	23	23	16	91	23	21	21	21	21	21	7
First Last AA of AA of	Sig Pep	20	20	22	22	15	15	22	70	20	20	20	20	9
First AA of	Sig Pep	1	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ	NO:Y	721	1084	722	1085	723	1086	724	725	1087	8801	726	1089	0601
5' NT of First AA of	Signal Pep	274	259	288	292	70	9	326	132	148	148	260	251	101
	Clone of Start Seq. Codon	274	259	288	292	70	99	326	132	148	148	260	251	
3' NT of	Clone Seq.	1266	1257	2803	2718	961	959	2181	2207	2206	5206	1242	628	903
5' NT of	Clone Seq.	-		-	1		-	-	-	1	1	-	1	29
	Total NT Seq.	1266	1257	2803	3302	961	959	2181	2207	2227	2214	1242	628	923
NT SEQ	АÄх	901	469	107	470	108	471	109	110	472	473	111	474	475
	Vector	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0
ATCC	Deposit No:Z and Date	203570 01/11/99	203570 01/11/99	PTA-848 10/13/99	PTA-848 10/13/99	209300 09/25/97	209300 09/25/97	203070 07/27/98	209965 06/11/98	209965 06/11/98	209965 06/11/98	PTA-868 10/26/99	PTA-868 10/26/99	PTA-868 10/26/99
	cDNA Clone ID	нронр03	нронр03	HDTBD53	HDTBD53	HDTBP04	HDTBP04	HDTBV77	нртр023	ното023	нртр023	HDTFE17	HDTFE17	HDTFE17
	Gene No.	96	96	26	62	86	86	66	100	100	100	101	101	101

Last AA of ORF	49	540	81	05	42	66	99	34	72
First AA of Secreted Portion	35	31	23	11	22	37	22	21	25
First Last AA of AA of Sig Sig Pep Pep	31	30	22	16	21	36	21	20	24
First AA of Sig Pep	1	1	1	1	-	1	1		
AA SEQ ID NO:Y	727	728	1001	729	730	731	732	733	734
5' NT of First AA of Signal Pep	386	808	515	LS	116	66	28	237	91
3' NT of 5' NT Clone of Start Seq. Codon	386	808	515	23	116	66	87	237	91
3' NT of Clone Seq.	712	3532	1115	370	1251	867	1558	989	2199
5' NT of Clone Seq.	1	2821	435	1	1	1	1	117	1
Total NT Seq.	712	3533	1145	370	2067	867	1558	1152	2199
× S B S ×	112	113	476	114	115	116	117	118	119
Vector	pCMVSport 2.0	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC Deposit No:Z and Date	209627 02/12/98	97923 03/07/97 209071 05/22/97	97923 03/07/97 209071 05/22/97	209300 09/25/97	97955 03/13/97 209074 05/22/97	209877 05/18/98	209603 01/29/98	97975 04/04/97 209081 05/29/97	PTA-1544 03/21/00
cDNA Clone ID	HDTGC73	HE2DE47	HE2DE47	HE2EN04	HE2FV03	HE2NV57	нЕ2РН36	нв6ЕU50	HE8DS15
Gene No.	102	103	103	104	105	106	107	108	109

Last AA of ORF	250	41	53	139	147	159	159	121	121	47	121	122
First AA of Secreted Portion	22	27	39	16	29	23	23	24	24	24	29	29
First Last AA of AA of Sig Sig Pep Pep	21	26	38	15	28	22	22	23	23	23	28	28
First AA of Sig Pep	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ ID NO:Y	735	736	737	738	739	740	1092	741	1093	742	743	1094
5' NT of First AA of Signal Pep	201	35	380	51	213	146	226	52	133	123	73	<i>L</i> 9
3' NT of 5' NT Clone of Start Seq. Codon	201	35	380	51	213	146	226	52	133	123	73	<i>L</i> 9
3' NT of Clone Seq.	1021	832	734	649	921	894	616	282	089	1336	662	803
5' NT of Clone Seq.	1	1	-	7	1	1	88	1	1	1	1	1
Total NT Seq.	1021	832	734	685	921	894	626	582	089	1336	662	802
SEQ NÖ:	120	121	122	123	124	125	477	126	478	127	128	479
Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pSport1	Uni-ZAP XR	Uni-ZAP XR
ATCC Deposit No:Z and Date	203570 01/11/99	209010 04/28/97 209085 05/29/97	. PTA-1543 03/21/2000	203069 07/27/98	203071 07/27/98	PTA-2076 06/09/00	PTA-2076 06/09/00	203071 07/27/98	203071 07/27/98	209563 12/18/97	209423 10/30/97	209423 10/30/97
cDNA Clone ID	HE8UB86	неэнүол	HE9NN84	HEBEJ18	HEEAQ11	HEEB105	HEEB105	HEGAN94	HEGAN94	неомо63	HEPAB80	HEPAB80
Gene No.	110	111	112	113	114	115	115	116	116	117	118	118

Last AA of	ORF	44	129	112	155	45	64	116	98	09	352	10	45
	Secreted Portion	18	28	28	28	41	22	22	43	23	21		18
First Last AA of AA of Sig Sig	Pep	17	27	27	27	40	21	21	42	22	20		17
First AA of Sig	Pep	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ ID	NO:Y	744	745	1095	1096	746	747	748	749	750	751	752	753
5' NT of First AA of Signal	Рер	198	25	62	23	09	161	199	232	655	47	487	4
	Codon	198	25	62	22	09	161	199	232	. 559	47		44
3' NT of Clone	Seq.	1689	1000	1052	1037	420	1569	1347	642	1323	1271	802	470
	Seq.	1	Ţ	30	1	-	1	-	1	60\$	1	352	1
Total	NT Seq.	1689	1000	1052	1037	420	1569	1347	642	1323	1271	802	470
N SEQ	ÿ×	129	130	480	481	131	132	133	134	135	136	137	138
	Vector	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	Uni-ZAP XR	Lambda ZAP II						
ATCC Deposit No:Z	and Date	209551 12/12/97	209965 06/11/98	209965 06/11/98	209965 06/11/98	209407 10/23/97	209877 05/18/98	209407 10/23/97	209277 09/18/97	209146 07/17/97	209463 11/14/97	209008 04/28/97 209084 05/29/97	209242 09/12/97
	cDNA Clone ID	HEQAK71	неосся	неосся	неосся	HERAR44	HETBR16	HFABH95	HFAEF57	HFAMB72	Н ЕССQ50	нғсев37	HFFAD59
	Gene No.	119	120	120	120	121	122	123	124	125	126	127	128

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Last AA of ORF	38	47	47	44	74	52	17	131	549	549	99	54	34
First AA of Secreted Portion	19	24	24	23	15	18		34	7.7	27	27	34	21
Last AA of Sig Pep	18	23	23	22	14	17		33	26	26	56	33	20
First AA of Sig Pep	11	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ ID No:Y	754	755	1097	756	757	758	1098	759	160	1099	1100	761	762
5' NT of First AA of Signal Pep	1019	24	74	20	137	25	15	414	185	249	185	178	158
5' NT of Start Codon	6101	24	74	20	137	25	.15	414	185	249	185	178	158
3' NT of Clone Seq.	1861	1408	1441	541	2407	540	539	2067	2213	2674	2207	532	740
5' NT of Clone Seq.	772	1	43	1	-	1	1	364	1	6\$	1	1	1
Total NT Sec.	1881	1408	1441	541	2407	540	539	2067	2213	2674	2207	532	762
Z B B S ×	139	140	482	141	142	143	483	144	145	484	485	146	147
Vector	Uni-ZAP XR	pSport1	pSport1	pSport1	Uni-ZAP XR	pSport1	pSport1	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC Deposit No:Z and Date	209225 08/28/97	PTA-846 10/13/99	PTA-846 10/13/99	209277 09/18/97	PTA-622 09/02/99	209277 09/18/97	209277 09/18/97	209626 02/12/98	209551 12/12/97	209551 12/12/97	209551	209242 09/12/97	209300 09/25/97
cDNA	HFGAD82	HFIIZ70	HFIIZ70	HEIUR10	HFKET18	HFOXA73	HFOXA73	HFPA071	HFPCX09	HFPCX09	HFPCX09	HFRAN90	HFTBM50
Gene	129	130	130	131	132	133	133	134	135	135	135	136	137

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Last AA of	ORF	519	89	26	79	162	85	162	99	47	79	46	292	377
First AA	Secreted Portion	21	23	32	25	25	23	27	26	19	19	24	17	24
Last AA of Sig	Pep	20	22	31	24	24	22	26	25	18	18	23	16	23
First AA of Sig	Pep	1	1	1	1	1	1	1	1	1	1		1	
AA SEQ U	NO:Y	763	. 764	765	992	191	168	692	1101	770	171	772	<i>ELL</i>	774
5' NT of First AA of Signal	Pep	93	547	114	213	152	13	141	148	98	204	273	87	14
5' NT of Start		93	547	114	213	152	13	141	148	98	204	273	87	14
3' NT of Clone	Seq.	1838	1103	1350	947	1633	1757	712	1347	1384	1715	1538	1276	1804
5' NT of Clone	Seq.	32	231	1	1	1	1	1	1	1	1	259	71	1
Total	NT Seq.	1839	1103	1350	947	1633	1757	712	1347	1384	1715	1538	1437	1816
NT SEQ	۶×	148	149	150	151	152	153	154	486	155	156	157	158	159
	Vector	Uni-ZAP XR	Uni-ZAP XR	pBluescript	Lambda ZAP II	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR						
ATCC Deposit No:Z	and Date	209782 04/20/98	209300 09/25/97	203105 08/13/98	209568 01/06/98	203071 07/27/98	209965 06/11/98	209423 10/30/97	209423 10/30/97	209782 04/20/98	209651 03/04/98	209011	209423 10/30/97	203648 02/09/99
	cDNA Clone ID	HFTDL56	HFTDZ36	HFVIC62	HFXAM76	HFXBL33	HFXGT26	HFXJU68	HFXJU68	HFXJX44	HFXKT05	HGBFO79	НСВНІЗ5	HGBIB74
	Gene No.	138	139	140	141	142	143	144	144	145	146	147	148	149

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	Last AA of	ORF	170	151	121	84	42	50	82	112	4	68
	First AA of	Secreted Portion	21	2	29	36	∞	28	21	30	34	23
Last	AA of AA of Sig Sig	Pep	20	-	28	35	7	27	20	29	33	22
First	AA of Sig	Pep	1	1	1	1	1	1	. 1	1	-	1
	SEQ E1	NO:Y	1102	1103	775	776	777	778	611	780	781	782
5' NT of First	AA of Signal	Pep	28	2	231	569	63	143	269	192	230	270
	of 5' NT Clone of Start		28		231	995	63	143	569	192	230	270
3	of Clone	Seq.	1821	1094	776	865	959	1155	1459	661	407	711
	of Clone	Seq.	1	1	1	229	1	1	1	-	1	8
	Total		1821	1094	176	865	959	1155	1459	661	407	711
ĮÄ) E	ÿ×	487	488	160	161	162	163	164	165	991	167
		Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	Uni-ZAP XR	Lambda ZAP II	Lambda ZAP II
	ATCC Deposit No:Z	and Date	203648 02/09/99	203648	209407 10/23/97	209179 07/24/97	209195 08/01/97	209368 10/16/97	PTA-322 07/09/99	97975 04/04/97 209081 05/29/97	97899 02/26/97 209045 05/15/97	97958 03/13/97 209072 05/22/97
		cDNA Clone ID	HGBIB74	HGBIB74	HGLAF75	HHEMA75	HHENK42	HHENV10	ннерм33	ниғнл59	HHGCG53	ннGCM76
		Gene No.	149	149	150	151	152	153	154	155	156	157

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Last AA of ORF	11	44	22	80\$	99	LL	51	44	130	122	327	99
First AA of Secreted Portion		41	20	28	22	29	61	28	2	2	24	14
Last AA of Sig Pep		40	19	27	21	28	81	72	1	1	23	13
First AA of Sig Pep	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ D NO:Y	1104	783	784	785	786	787	788	789	1105	1106	790	791
5' NT of First AA of Signal Pep	270	107	71	183	06	74	557	291	95	350	232	348
S' NT of Start Codon	270	107	71	183	06	74	557	291			232	348
	711	1050	488	2152	1113	1555	1061	1532	1614	1087	1272	1064
5' NT 3' NT of of Clone Clone Seq. Seq.	∞	1	-	141	1	1	454	1	1020	491	93	306
Total NT Seq.	711	1050	488	2152	1113	1555	1061	1532	1614	1087	1559	1064
X S B S X	489	168	169	170	171	172	173	174	490	491	175	176
Vector	Lambda ZAP II	Lambda ZAP II	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pBluescript SK-	pBluescript SK-	pBluescript SK-	pBluescript SK-	pBluescript SK-	pBluescript SK-	pCMVSport 3.0
ATCC Deposit No:Z and Date	97958 03/13/97 209072 05/22/97	209346 10/09/97	209877 05/18/98	209746 04/07/98	209346 10/09/97	209119 06/12/97	209146 07/17/97	PTA-843 10/13/99	PTA-843 10/13/99	PTA-843 10/13/99	209877 05/18/98	209407 10/23/97
cDNA Clone ID	нндсм76	HHGDW43	HHPEC09	HHPEN62	HHSDX28	HJABB94	HJABX32	HJACG30	HJACG30	HJACG30	HJBCY35	HJMBN89
Gene No.	157	158	159	160	161	162	163	164	164	164	165	166

Last	AA of ORF	16	47	243	243	08	301	154	275	438	57	43	107	107
Fir	of Secreted Portion	30	17	18	18	24	56	56	56	31	30	31	42	42
Last AA of	Sig Pep	29	16	17	17	23	25	25	25	30	29	30	41	41
. . .	Sig Pep	1	1	1	1	1	1	1	1	1	1	1	-	1
AA SEQ	MO:Y	792	793	794	1107	795	962	1108	161	798	1109	799	800	1110
5' NT of First AA of	Signal Pep	09	274	11	69	27	38	35	229	501	197	243	208	508
	Seq. Codon	09	274	11	69	27	38	35	229	501	197	243	208	508
3' NT of	Clone Seq.	1231	1238	1189	1191	496	3153	1626	1517	2496	2351	549	1001	1001
5° NT of	Clone Seq.	1	45	7	1	1	1	1	30	1	1		270	270
	Total NT Seq.	1231	1238	1189	1191	496	3153	1626	1523	2496	2351	549	1001	1001
NT SEQ	≅ë×	177	178	179	492	180	181	493	182	183	494	184	185	495
	Vector	Uni-ZAP XR	pCMVSport 2.0	pCMVSport 2.0	· pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0
ATCC	Deposit No:Z and Date	209641 02/25/98	209603 01/29/98	209683 03/20/98	209683 03/20/98	209346 10/09/97	209346 10/09/97	209346 10/09/97	209568 01/06/98	209627 02/12/98	209627 02/12/98	209300 09/25/97	PTA-849 10/13/99	PTA-849 10/13/99
	cDNA Clone ID	HJPAD75	HKAB184	HKABZ65	HKABZ65	HKACB56	HKACD58	HKACD58	НКАDQ91	HKAEV06	HKAEV06	HKAFK41	HKAFT66	HKAFT66
	Gene No.	167	168	169	169	170	171	171	172	173	173	174	175	175

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Last AA of ORF	37	234	46	470	442	309	243	93	47	260	148	95	9/
First AA of Secreted	Portion	31	27	16	61	2	2	27	27	34	34	20	33
Last AA of Sig Pep	•	30	26	15	18	1	1	26	26	33	33	19	32
First AA of Sig Pep	1	-	1	1	1	1	1	1	1	1	1	1	1
AA SEQ D No:Y	1111	801	1112	802	1113	1114	1115	803	1116	804	1117	805	806
5' NT of First AA of Signal Pep	234	178	30	64	41	3	3	313	57	53	55	130	43
5' NT of Start Codon	234	178	30	64	41			313	57	53	55	130	43
3' NT of Clone Seq.	,	1142	417	2238	1906	1487	1525	1021	1311	1052	1050	1439	1215
5' NT of Clone Seq.	1	1038	1	1	1	-	1	1	1	1	1	1	1
Total NT	Seq. 669	1142	417	2238	1949	1487	1525	1021	1311	1052	1050	1492	1215
SEQ H	X 496	186	497	187	498	499	200	188	501	681	502	190	191
	Vector pCMVSport	2.0 pCMVSport 1	pCMVSport 1	ZAP Express	ZAP Express	ZAP Express	ZAP Express	pSport1	pSport1	pSport1	pSportl	pBluescript	pBluescript
ATCC Deposit No:Z and Date	PTA-849	209651 03/04/98	209651 03/04/98	209782 04/20/98	209782 04/20/98	209782 04/20/98	209782 04/20/98	209853 05/07/98	209853 05/07/98	209877 05/18/98	209877 05/18/98	209603 01/29/98	209463 11/14/97
cDNA	Clone ID HKAFT66	HKB1E57	HKB1E57	НКҒВС53	НКҒВС53	НКҒВС53	НКҒВС53	HKGC027	HKGC027	HKGDL36	HKGDL36	HKISB57	HKIYP40
Gene	No. 175	176	176	177	177	177	177	178	178	179	179	180	181

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Last	AA of ORF	69	130	41	113	275	191	348	44	116	50	206	58	28
Fírst AA	of Secreted Portion	56	21	22	21	22	61	24	22	20	36	30	20	18
First Last AA of AA of	Sig Pep	25	20	21	20	21	18	23	21	61	35	29	19	17
First AA of	Sig Pep	1	1	1	1	1	1	1	ı	1	1	1	1	1
AA SEQ	NO:Y	807	808	808	810	811	812	813	814	815	816	817	818	819
5' NT of First AA of	Signal Pep	20	82	202	368	43	520	66	30	224	186	249	205	74
5° NT	of Start Codon	20	82	202	368	43	520	66	30	224	186	249	205	74
3' NT of	Clone Seq.	1543	954	1794	1256	686	2572	1488	704	613	1022	1766	913	770
5' NT 3' NT of	Clone Seq.	1	1		208	-	427	П	-	1	1		1	1
	Total NT Seq.	1543	954	1794	1262	686	2572	1488	704	613	1022	99/1	913	0//
NT SEQ	ВÄХ	192	193	194	195	196	197	198	199	200	201	202	203	204
	Vector	pBluescript	pBluescript	pBluescript	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	Uni-ZAP XR	Uni-ZAP XR	pCMVSport 1	pCMVSport 1	Lambda ZAP II	Uni-ZAP XR
ATCC	Deposit No:Z and Date	209511 12/03/97	209236 09/04/97	209463 11/14/97	209628 02/12/98	PTA-1544 03/21/00	203027 06/26/98	203071 07/27/98	209746 04/07/98	209126 06/19/97	203071 07/27/98	203517 12/10/98	209551 12/12/97	209243 09/12/97
	cDNA Clone ID	HKMLK53	HKMLM11	HKMMW74	HLDON23	HLDOW79	HLDQR62	нгролу	HLHAL68	HLHFP03	HLBD68	нгісо90	нгорн79	HLTDV50
	Gene No.	182	183	184	185	186	187	188	189	190	191	192	193	194

	Last AA of	ORF	42	75	62	65	299	187	140	354	338	58	46	222	215
<u> </u>			4	, ,	5)	2	1	1	3	3	-	4	2	2
	First AA of	Secreted Portion	20	35	27	27	2	16	25	22	26	37	17	19	19
	۹,	Pep	19	34	56	26	I	15	24	21	25	96	16	18	18
First	AA of Sig	Pep	1	ī	1	1	1	1	1	1	1	1	1	1	1
AA S) E	NO:Y	820	821	822	1118	1119	823	824	825	826	<i>L</i> 78	828	678	1120
5' NT of First	AA of Signal	Pep	155	5	226	226	3	436	326	212	38	551	92	190	205
	5' NT of Start	Codon	155	5	226	226		436	326	212	38	155	92	190	205
3° NT	of Clone	Seq.	843	2286	1170	647	1209	997	1167	1311	1892	646	312	1237	997
T	of Clone	Seq.	-	-	1		870	246	304	-	-	1	_		74
	Total	NT Seq.	843	2286	1240	647	1321	266	1167	1338	1892	646	312	1237	266
F	SE DE DE	ÿ×	205	206	207	503	504	208	209	210	211	212	213	214	505
		Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pSport1	pSport1	pSport1
	ATCC Deposit No:Z	and Date	97979 03/27/97	209782 04/20/98	PTA-2076 06/09/00	PTA-2076 06/09/00	PTA-2076 06/09/00	209626 02/12/98	209651 03/04/98	209511 12/03/97	209651 03/04/98	209126 06/19/97	203071 07/27/98	209022 05/08/97	209022 05/08/97
		cDNA Clone ID	HLTE125	HLTHR66	HLTIP94	HLTIP94	HLTIP94	HLWAA17	HLWAD77	HLWA022	HLWAY54	HLWCF05	HLYAC95	HLYAZ61	HLYAZ61
		Gene No.	195	196	197	197	197	198	199	200	201	202	203	204	204

Last AA of	ORF	152	713	77	340	306	64	44	82	54	221	33	62	76
First AA	ed on	25	17	16	27	27	28	33	44	29	35	70	28	14
First Last AA of AA of Sig		24	16	15	56	56	27	32	43	28	34	19	27	13
First AA of Sig	Pep	1	1	1	1	1	1	1	1	1	1		-	1
AA SEQ	NO:Y	830	831	1121	832	1122	833	834	835	836	837	838	839	840
5' NT of First AA of Signal		191	491	115	4	3	175	273	180	50	34	332	92	49
3' NT of 5' NT Clone of Start	Codon	161	491	115	4	3	175	273	180	20	34	332	92	49
3' NT of	Seq.	864	3194	437	1258	1084	883	1465	974	1010	1369	596	611	1420
5° NT of		1	1	1	1	-		1	-		28	-	-	1
Total	NT Seq.	864	3194	437	1258	1084	883	1465	974	1010	1369	596	629	1420
NT SEQ	ë ë ×	215	216	206	217	507	218	219	220	221	222	223	224	225
	Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Lambda ZAP II	Lambda ZAP II	Lambda ZAP II	Lambda ZAP II	Uni-ZAP XR
ATCC Denosit No.2	and Date	209368 10/16/97	209139 07/03/97	209139 07/03/97	PTA-2075 06/09/00	PTA-2075 06/09/00	209628 02/12/98	209368 10/16/97	209563 12/18/97	209853 05/07/98	209368 10/16/97	209243 09/12/97	209243 09/12/97	209563 12/18/97
	cDNA Clone ID	HMADK33	HMADU73	HMADU73	HMAMI15	HMAMI15	HMCFY13	HMDAB56	HMDAQ29	HMECK83	HMEED18	HMEFT54	HMEGF92	HMIAL37
	Gene No.	202	206	206	207	207	208	209	210	211	212	213	214	215

Last	AA of ORF	49	90	406	406	64	64	26	20	62	37	103	103	73
First AA	of Secreted Portion	29	16	33	33	27	27	7	2	35	18	24	24	23
Last AA of	Sig Pep	28	15	32	32	26	26	9	1	34	17	23	23	22
First AA of	Sig Pep	1	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ	AÖ.Y	841	842	843	1123	844	1124	1125	1126	845	846	847	1127	848
5' NT of First AA of	Signal Pep	221	142	157	192	531	528	565	2	120	28	134	162	133
s. NT	of Start Codon	221	142	157	192	531	528	565		120	28	134	162	133
3' NT of	Clone Seq.	921	822	1871	1897	2497	1776	784	427	1217	1283	1402	616	1417
5' NT of	Clone Seq.	99	-	-	37	1	1	1	275	1	1	1	30	1
	Total NT Seq.	921	822	1871	1914	2497	1776	784	669	1217	1283	1402	616	1417
NT SEQ	ДÄХ	226	227	228	508	229	509	510	511	230	231	232	512	233
	Vector	pSport1	pSport1	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC	Deposit No:Z and Date	209346 10/09/97	209368 10/16/97	209022 05/08/97	209022 05/08/97	PTA-842 10/13/99	PTA-842 10/13/99	PTA-842 10/13/99	PTA-842 10/13/99	209368 10/16/97	209324 10/02/97	PTA-2070 06/09/00	PTA-2070 06/09/00	203105 08/13/98
	cDNA Clone ID	HMKCG09	HMMAH60	нморт36	нморт36	HMSDL37	HMSDL37	HMSDL37	HMSDL37	HMSF126	HMSFS21	HMSHS36	HMSHS36	HMSKC04
	Gene No.	216	217	218	218	219	219	219	219	220	221	222	222	223

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Last	AA of ORF	84	139	42	121	30	18	490	188	71	89	62	233	99
First AA		20	44	31	28	91	01	22	22	22	13	33	35	22
Last AA of	Sig Pep	19	43	30	27	15	6	21	21	21	12	32	34	21
First AA of	Sig Pep	1	1	1	1	1	1	1	-	1	1	-	1	1
AA SEO	NO:Y	849	850	851	852	853	1128	854	1129	855	856	857	828	829
5' NT of First AA of	Signal Pep	306	34	124	72	367	129	42	42	213	488	170	228	98
S' NT	Clone of Start Seq. Codon	306	34	124	72	367		42	42	213	488	170	228	98
3' NT of	Clone Seq.	1173	529	754	1346	556	929	1974	1976	1079	2058	456	1212	616
5' NT of	Clone Seq.	1	1	105	-	1	1	1	1	1	209	-	28	1
	Total NT	1173	529	1146	1346	556	929	1974	2027	1079	2103	456	1212	616
NT	A Š A	234	235	236	237	238	513	239	514	240	241	242	243	244
	Vector	pCMVSport	pSport1	Uni-ZAP XR	pBluescript	pBluescript	Uni-ZAP XR							
ATCC	Deposit No:Z and Date	209551	209628 02/12/98	209126 06/19/97	209368 10/16/97	209147 07/17/97	209147 07/17/97	203105 08/13/98	203105 08/13/98	209346 10/09/97	203027 06/26/98	209126 06/19/97	209628 02/12/98	209463 11/14/97
	cDNA	HMTAD67	HMVBS81	HMWDC28	HMWFT65	HMWFY10	HMWFY10	HMWGY65	HIMWGY65	HNEEE24	HNFFC43	HNFIU96	HNFIY77	HNFJF07
	Gene	224	225	226	227	228	228	229	229	230	231	232	233	234

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Last AA of ORF		<i>L</i> 9	33	4	06	40	36	70	46	74	53	82	57	57
First AA of Secreted	Portion	31	20	33	24	32	17	22	37	15	25	28	35	35
Last AA of Sig Peo	1	30	19	32	23	31	16	21	36	14	24	27	34	34
First AA of Sig Pep	J.	1	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ ID		860	861	862	863	864	865	866	867	868	869	870	871	1130
5' NT of First AA of Signal Pep	1. ·	275	50	86	108	178	135	221	11	321	436	388	27	27
3' NT of 5' NT Clone of Start Sec. Codon		275	50	86	108	178	135	221	11	321	436	388	27	27
3' NT of Clone		575	203	491	236	427	962	527	1037	985	925	841	2128	774
5' NT of Clone Sea	·koo	1	1	1	1	1	1	1	1	1	1	1	1	1
Total NT	Seq.	275	703	491	536	427	96L	527	1037	985	. 925	841	2128	774
N S B S	X.	245	246	247	248	249	250	251	252	253	254	255	256	515
	Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC Deposit No:Z	ally Dale	97976 04/04/97	209243 09/12/97	209299 09/25/97	209407 10/23/97	97976 04/04/97	209236 09/04/97	209243 09/12/97	209368 10/16/97	209603 01/29/98	203648 02/09/99	203648 02/09/99	PTA-847 10/13/99	PTA-847 10/13/99
AZ C	Clone ID	HNFJH45	HNGAP93	HNGE029	HNGFR31	HNGIH43	HNGIJ31	HNGIQ46	HNGJE50	HNGJP69	HNGKN89	HNGND37	HNGOI12	HNGOI12
one.	No.	235	236	237	238	239	240	241	242	243	244	245	246	246

Last AA of ORF	93		54	36	36	28	34	81	80	53	80	40	320	172
First AA of Secreted	Portion 26	}	42	23	23	7	4	35	29	21	21	34	36	36
Last AA of Sig Pep	25		41	22	22	9	3	34	28	70	70	33	35	35
First AA of Sig Pep			-	-		1	1	1	1	1	1	1	1	-
AA SEQ D NO:Y	1131		872	873	1132	1133	1134	874	875	876	877	878	628	1135
5' NT of First AA of Signal Pep	596	3	328	52	28	166	331	57	38	40	12	291	28	32
3' NT of S' NT Clone of Start Seq. Codon			328	52	28			57	38	40	12	291	28	32
3' NT of Clone Seq.	1396	2	905	2642	1654	447	149	748	297	1894	1355	940	1382	1397
5' NT of Clone Seq.	• -		1	1	1	1	1	1	1	1	1	-	1	1
Total NT	Seq.	222	905	2642	1654	447	641	748	297	1894	1355	940	1382	1397
SEQ BO NO:	× 516	2	257	258	517	518	519	259	260	261	262	263	264	520
	Vector IIni-ZAP XR	OIII-EAL AXX	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pCMVSport 3.0	pCMVSport 3.0
ATCC Deposit No:Z and Date	PTA-847	10/13/99	209180 07/24/97	PTA-844 10/13/99	PTA-844 10/13/99	PTA-844 10/13/99	PTA-844 10/13/99	209628 02/12/98	209683 03/20/98	PTA-623 09/02/99	PTA-1543 03/21/00	203570 01/11/99	209563 12/18/97	209563 12/18/97
cDNA	7/2	11100111	HINHAH01	HNHE142	HNHEI42	HNHE142	HNHEI42	HINHEU93	HNHFM14	HINHINB29	HNHOD46	HNHPD10	HNTB126	HNTB126
Gene	No.	2+7	247	248	248	248	248	249	250	251	252	253	254	254

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Last	AA OI ORF	131	58	115	402	121	92	49	41	20	35	159	148
Fir	Secreted Portion	98	LZ	24	31	59	34	34	74	15	27	2	19
	Sig Pep	35	26	23	30	28	33	33	23	14	26	1	18
First AA of	Sig Pep	1	1	1	1	1	1	1	1	1	-	1	1
AA SEQ	MO:Y	1136	880	881	882	1137	883	1138	884	885	988	887	1139
5' NT of First AA of	Signal Pep	16	210	100	111	23	307	306	46	251	434	1	27
	Seq. Codon	16	210	100	111	27	307	306	46	251			27
3. NT of	Clone Seq.	1368	1365	791	2163	1763	2087	1114	830	755	1939	1126	1124
S' NT of	Clone Seq.	1	134	71	830	1	1	1	1	1	294	1	1
E	NT Seq.	1368	1365	791	2163	1763	2087	1274	830	755	1939	1126	1124
NT SEQ	₽Ä×	521	265	266	267	522	268	523	269	270	271	272	524
	Vector	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pSport1	pSport1	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC	Deposit No:Z and Date	209563 12/18/97	209423 10/30/97	20932 4 10/02/97	PTA-1544 03/21/00	PTA-1544 03/21/00	209782 04/20/98	209782 04/20/98	203069 07/27/98	209244 09/12/97	209012 04/28/97 209089 06/05/97	203570 01/11/99	203570 01/11/99
	cDNA Clone ID	HNTB126	HNTBI57	HNTBL27	HNTCE26	HNTCE26	HNTNI01	HNTNI01	HODDF13	HODDN65	НОДДИ НОДДИ НО	HODFN71	HODFN71
	Gene No.	254	255	256	257	257	258	258	259	260	261	262	797

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Last AA of	ORF	180	484	484	266	5	84	410	115	92	368	73	72	129
First AA of	Secreted Portion	14	25	25	25		2	21	21	10	21	14	14	19
Last AA of Sig	Pep	13	24	24	24		1	20	20	6	70	13	13	18
First AA of Sig	Pep	1		1	1	1	1	1	1	1	1	1	1	-
AA SEQ D	NO:Y	888	889	1140	.1141	1142	1143	890	1144	1145	1146	891	1147	892
5' NT of First AA of Signal	Pep	49	49	48	78	724	123	83	83	1225	129	62	155	18
5" NT of Start	Codon	64	49	48	78			83	83		129	79	155	18
3' NT of Clone	Seq.	2657	2410	2409	9/8	1586	1011	2131	427	1500	1234	7194	3095	1491
5' NT of Clone	Seq.	1	1	1	1	1	873	9	1	1	337	1	1	1
Total	NT Seq.	2657	2410	2409	928	1586	1011	2131	427	1500	1234	2794	3095	1491
NT SEQ D	ÿ×	273	274	525	526	527	528	275	529	530	531	276	532	277
	Vector	Uni-ZAP XR	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0
ATCC Deposit No:Z	and Date	203517 12/10/98	PTA-848 10/13/99	PTA-623 09/02/99	PTA-623 09/02/99	PTA-848 10/13/99								
	cDNA Clone ID	HOEFV61	НОҒМQ33	ноғм033	НОFMQ33	НО FMQ33	НО FMQ33	HOFMT75	HOFMT75	HOFMT75	HOFMT75	HOFNC14	HOFNC14	НОБОС73
	Gene No.	263	264	264	264	264	264	265	265	265	265	266	266	267

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Last AA of ORF	19	14	9	246	2	199	211	211	<u>2</u>	161	325	26	40
First AA of Secreted	Portion 19	5		30		26	26	26	22	25	2	31	19
Last AA of Sig Pep	18	4		29		25	25	25	21	24	1	30	18
First AA of Sig Pep	-	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ ID NO:Y	1148	1149	1150	893	1151	894	895	1152	968	1153	1154	897	868
5' NT of First AA of Signal Pep	23	127	142	514	1455	232	136	144	361	102	SS	68	1076
S' NT 3' NT of Firs of Firs of Clone Clone of Start Signal Seq. Codon Pep	23		142	514		232	136	144	361	102		68	9/01
3' NT of Clone Seq.	1395	270	2324	1409	1697	1188	1623	1632	3530	285	1942	1145	2214
5' NT of Clone Seq.	-	-	662	310	144	1	1	17	1	2	1339	_	985
Total NT	Seq. 1395	270	2324	1409	1697	1188	1623	1637	3530	585	4344	1145	2214
SEQ SEQ	X 533	534	535	278	536	279	780	537	281	538	539	282	283
	Vector pCMVSport	pCMVSport	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	pBluescript SK-	pBluescript SK-	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC Deposit No:Z and Date	PTA-848	PTA-848 10/13/99	PTA-848 10/13/99	PTA-848 10/13/99	PTA-848 10/13/99	209603 01/29/98	209138 07/03/97	209138 07/03/97	PTA-845 10/13/99	PTA-845 10/13/99	PTA-845 10/13/99	209551 12/12/97	209423 10/30/97
cDNA	Clone ID HOFOC73	НОГОС73	НОГОС73	НОССК63	носск63	HOHBY12	HONAH29	HONAH29	НООВ182	НООВ182	НООВ182	HOSBY40	HOSD125
Gene	No. 267	267	267	268	268	269	270	270	271	271	271	272	273

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Last AA of ORF	40	624	61	<i>L</i> 96	119	131	131	26
First AA of Secreted Portion	19	31	33	52	28	30	30	19
First Last AA of AA of Sig Sig Pep Pep	18	30	32	51	72	29	29	18
First AA of Sig Pep	1	1	1	1	1	1	T	1
AA SEQ ID NO:Y	1155	668	1156	006	106	905	1157	903
5' NT of First AA of Signal Pep	146	99	477	208	98	51	510	203
3' NT of S' NT Clone of Start Seq. Codon	146	99	477	208	98	51	510	203
3' NT of Clone Seq.	1258	1747	1747	4693	599	8/6	1442	625
5' NT of Clone Seq.	1	290	288	-	1	-	455	
Total NT Seq.	1258	2527	2527	4712	599	978	2361	625
SEQ X	540	284	541	285	286	287	542	288
Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pBluescript SK-	pBluescript SK-	pBluescript SK-	Uni-ZAP XR
ATCC Deposit No:Z and Date	209423 10/30/97	97957 03/13/97 209073 05/22/97	97957 03/13/97 209073 05/22/97	209086 05/29/97	97977 04/04/97 209082 05/29/97	209012 04/28/97 209089 06/05/97	209012 04/28/97 209089 06/05/97	209244 09/12/97
cDNA Clone ID	HOSDJ25	HOSFD58	HOSFD58	HOUCQ17	HPBCU51	HPDDC77	HPDDC77	HPEAD48
Gene No.	273	274	274	275	276	277	277	278

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Last	AA of ORF	41	15	211	173	53	48	48	10	4	201	201	420	392
First AA	ot Secreted Portion	16	12	19	19	32	19	19			26	26	30	30
Last AA of	Sig Pep	15	11	18	18	31	18	18			25	25	29	29
	Sig Pep	1	1	1	-	1	1	1	1	1	1	1	1	1
AA SEQ	NO:Y	904	905	906	1158	907	806	1159	1160	1161	906	1162	910	1163
5' NT of First AA of	Signal Pep	51	79	128	127	236	126	119	696	509	64	58	62	70
	Clone of Start Seq. Codon	51	62	128	127	236	126	119		509	64	58	62	70
3' NT of	Clone Seq.	813	265	1739	1739	1677	2648	538	1346	912	1084	1083	2072	1775
	Clone Seq.	1	1	1		-		-	1		1	-	1	1038
	Total NT Seq.	813	597	1739	1739	1677	2648	538	1346	912	1084	1177	2072	1775
NT SEQ	ВÄ×	289	290	291	543	292	293	544	545	546	294	547	295	548
	Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR					
ATCC	Deposit No:Z and Date	209244 09/12/97	209241 09/12/97	209563 12/18/97	209563 12/18/97	209889 05/22/98	PTA-855 10/18/99	PTA-855 10/18/99	PTA-855 10/18/99	PTA-855 10/18/99	209628 02/12/98	209628 02/12/98	209195 08/01/97	209195 08/01/97
	cDNA Clone ID	HPEAD79	HPEBE79	HPIBO15	HPB015	HPJB133	HPJBK12	HPJBK12	HPJBK12	HPJBK12	HPMDK28	HPMDK28	HPRAL78	HPRAL78
	Gene No.	279	280	281	281	282	283	283	283	283	284	284	285	285

Last	AA of ORF	63	387	69	59	80	44	25	52	98	78	54	54
First AA	of Secreted Portion	43	31	27	23	27	26	31	31	2	28	19	22
First Last AA of AA of	Sig Pep	42	30	26	22	26	25	30	30	1	27	18	21
First AA of	Sig Pep	1	1	1	_	1	1	1	1	1	-	1	-
A.A. SEQ	, ED NO:Y	1164	911	1165	912	913	914	915	1166	1167	916	917	1168
5' NT of First AA of	Signal Pep	148	94	404	127	318	80	468	474	178	88	149	149
S' NT		148	94	404	127	318	80	468	474		88	149	149
3' NT of	Clone Seq.	998	2543	2032	741	803	819	1414	891	501	323	1340	1340
5' NT of	Clone Seq.	128	1245	275	1	215	1	1	1	120	-	-	1
	Total NT Seq.	998	.2543	2052	741	803	819	1414	891	501	323	1340	1340
NT SEO	́Вё×	549	296	550	297	298	299	300	551	552	301	302	553
	Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pBluescript	pBluescript	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC	Deposit No:Z and Date	209195 08/01/97	209852 05/07/98	209852 05/07/98	209244 09/12/97	209628 02/12/98	209244 09/12/97	PTA-843 10/13/99	PTA-843 10/13/99	PTA-843 10/13/99	209007 04/28/97 209083 05/29/97	209852 05/07/98	209852 05/07/98
	cDNA Clone ID	HPRAL78	HPRBC80	HPRBC80	HPRSB76	HPTVX32	HPVAB94	HPWAY46	HPWAY46	HPWAY46	HPWAZ95	HPWDJ42	HPWDJ42
	Gene No.	285	286	286	287	288	289	290	290	290	291	292	292

														
Last	AA of ORF	47	47	159	102	102	53	23	472	472	178	329	199	2
First A.A.	of Secreted Portion	19	19	15	28	28	41	41	25	25	2	28	39	
First Last AA of AA of	Sig Pep	18	18	14	27	27	40	40	24	24		L7	38	
First AA of	Sig Pep	. 1	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ	NO:Y	1169	918	919	920	1170	921	1171	922	1172	1173	923	1174	1175
5' NT of First AA of	Signal Pep	161	34	35	144	130	252	252	132	66	1	30	30	11
S' NT	Clone of Start Seq. Codon	161	34	35	144	130	252	252	132	66		30	30	
3' NT of	Clone Seq.	813	1676	1747	1251	1237	1539	1453	2077	1863	1134	2108	979	152
5' NT of	Clone Seq.	-	1	1	1	-	24	24	1	8	1	1	8	1
	Total NT Seq.	813	9/91	1747	1251	1237	1539	1891	2077	1863	1134	2108	979	152
NT SEQ	ЯÄ×	554	303	304	305	555	306	556	307	557	558	308	559	260
	Vector	Uni-ZAP XR	pBluescript	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC	Deposit No:Z and Date	209852 05/07/98	209511 12/03/97	209651 03/04/98	209889 05/22/98	209889 05/22/98	209852 05/07/98	209852 05/07/98	209878 05/18/98	209878 05/18/98	209878 05/18/98	PTA-841 10/13/99	PTA-841 10/13/99	PTA-841 10/13/99
	cDNA Clone ID	HPWDJ42	HPZAB47	HRAAB15	HRABA80	HRABA80	HRACD15	HRACD15	HRACJ35	HRACJ35	HRACJ35	HRGBL78	HRGBL78	HRGBL78
	Gene No.	292	293	294	295	295	296	296	297	297	297	298	298	298

Last AA of	ORF	32	379	283	286	48	85	100	142	45	57	262	399	305
First AA	Secreted Portion	11	31	16	16	23	21	22	27	30	25	19	20	22
Last AA of Sig	Pep	10	30	15	15	22	20	21	26	29	24	81	61	21
First AA of Sig	Pep	1	-	1	1	1	1	1	1	1	1	1	1	1
AA SEQ	NO:Y	1176	924	1177	1178	925	926	726	928	1179	929	930	931	1180
5' NT of First AA of Signal	Pep	1048	10	31	247	122	155	104	142	122	98	299	09	126
5' NT			10	31	247	122	155	104	142	122	86	299	09	126
3' NT of	Seq.	1760	1146	880	1106	1998	191	009	970	646	1388	1537	1782	1590
5' NT of	Seq.	127	224	1	224	1	2		106	1	1	92	-	96
Total	NT Seq.	1760	1146	088	9011	8661	773	009	026	646	1388	1537	1782	1590
SEQ	ığ×	561	309	562	563	310	311	312	313	564	314	315	316	565
	Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC	and Date	PTA-841 10/13/99	PTA-2069 06/09/00	PTA-2069 06/09/00	PTA-2069 06/09/00	203499 12/01/98	209124 06/19/97	209651 03/04/98	209126 06/19/97	209126 06/19/97	209463 11/14/97	209746 04/07/98	209603 01/29/98	209603 01/29/98
~	cDNA Clone ID	HRGBL78	HROAJ39	HROAJ39	HROAJ39	HROBD68	HSAVD46	HSAVH65	HSAWD74	HSAWD74	HSAWZ41	HSDAJ46	HSDEK49	HSDEK49
	Gene No.	298	299	299	299	300	301	302	303	303	304	305	306	306

														
Last	AA of ORF	122	97	223	72	70	152	217	219	219	135	121	181	6
First AA	of Secreted Portion	42	42	21	20	20	21	34	. 36	36	18	18	19	
First Last AA of AA of	Sig Pep	41	41	20	19	19	20	33	35	35	11	17	81	
First AA of	Sig Pep	1	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ	NO:Y	932	1181	933	1182	934	935	936	1183	1184	937	1185	938	939
5' NT of First AA of	Signal Pep	58	99	66	66	118	247	84	27	78	16	22	160	44
S' NT	Clone of Start Seq. Codon	58	99	66	66	118	247	84	27	78	16	22	160	44
3° NT of	Clone Seq.	795	1540	1179	1179	1742	1443	2523	2467	2523	608	819	1151	898
S' NT of	Clone Seq.	1	1	23	1	1	1	1	-	1		-	-	1
	Total NT Seq.	795	1540	1205	1179	1742	1443	2541	2467	2541	608	819	1151	898
NT SEQ	日 S X	317	999	318	567	319	320	321	268	995	322	570	323	324
	Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pBluescript	pBluescript	pBluescript	Uni-ZAP XR						
ATCC	Deposit No:Z and Date	209852 05/07/98	209852 05/07/98	203648 02/09/99	203648 02/09/99	209551 12/12/97	203081 07/30/98	PTA-884 10/28/99	PTA-884 10/28/99	PTA-884 10/28/99	209145 07/17/97	209145	209324 10/02/97	209346 10/09/97
	cDNA Clone ID	HSDEZ20	HSDEZ20	HSDFJ26	HSDFJ26	HSDFW45	HSDJA15	HSDJL42	HSDJL42	HSDJL42	HSDSB09	HSDSB09	HSDSE75	HSFAM31
	Gene No.	307	307	308	308	309	310	311	311	311	312	312	313	314

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Last	AA of ORF	256	58	28	286	171	950	509	554	260	23	247
First AA		17	16	23	28	32	25	22	22	24	19	28
Last AA of	Sig Pep	16	15	22	27	31	24	21	21	23	18	27
First AA of	Sig Pep	1	1	1	1	1	1	П	1	1	1	1
AA	NO:Y	940	1186	941	942	1187	943	1188	1189	944	1190	945
5' NT of First AA of	Signal Pep	117	150	8	49	393	786	127	12	353	537	64
Z. V.		117	150	8	49	393	786	127	12	353	537	64
3' NT	Clone Seq.	1410	1450	1303	1120	1250	4412	1792	1673	1432	2084	696
S' NT 3' NT	Clone Seq.	-	1	1	219	223	-	134	-	151	335	1
	Total NT Seq.	1410	1450	1303	1251	1250	4412	1792	1673	1907	2084	696
NT	N N N N N	325	571	326	327	572	328	573	574	329	575	330
	Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pBluescript	pBluescript	pBluescript					
ATC	Deposit No:Z and Date	209580 01/14/98	209580 01/14/98	209551 12/12/97	209009 04/28/97	209009 04/28/97	PTA-322 07/09/99	PTA-322 07/09/99	PTA-322 07/09/99	97977 04/04/97 209082 05/29/97	97977 04/04/97 209082 05/29/97	209346 10/09/97
	cDNA Clone ID	HSICV24	HSICV24	HSIDJ81	HSKCP69	HSKCP69	HSKDA27	HSKDA27	HSKDA27	HSKGN81	HSKGN81	HSKHZ81
	Gene No.	315	315	316	317	317	318	318	318	319	319	320

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Last	AA of ORF		247	42	42	9	35	55	25	62	218	218
First AA	of Secreted	Portion	28	17	17		20	19	81	20	21	20
First Last AA of AA of	Sig Pep		27	. 16	16		19	18	17	19	20	19
First AA of	Sig Pep	•	1	1	1	1	1	1	1	1	1	1
AA SEQ			1191	946	1192	1193	947	948	1194	949	026	1195
5' NT of First AA of	Signal Pep	•	27	114	206	1331	220	225	232	130	87	91
S' NT	Clone of Start Signal Seq. Codon Pep		22	114	206		220	225	232	130	87	91
3' NT of	Clone Sea.	1	296	2126	1083	1904	861	285	720	654	931	971
S' NT 3' NT of	o .	1	1	1	1	1	1	1	1	1	_	13
	Total NT	Seq.	886	2126	£801	1904	861	282	720	657	931	971
NT SEQ	Ή̈́	×	919	331	577	578	332	333	579	334	335	580
		Vector	pBluescript	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC	Deposit No:Z		209346 10/09/97	PTA-855 10/18/99	PTA-855 10/18/99	PTA-855 10/18/99	209139 07/03/97	209300 09/25/97	209300 09/25/97	209641 02/25/98	97974 04/04/97 209080 05/29/97	97974 04/04/97 209080 05/29/97
		Clone ID	HSKHZ81	HSLJG37	HSLJG37	HSLJG37	HSNAD72	HSNMC45	HSNMC45	НЅQСМ10	HSQEO84	HSQE084
	Gene	Š.	320	321	321	321	322	323	323	324	325	325

Last AA of ORF	99	78	41	909	909	99	88	06	45	305	305
First AA of Secreted Portion	21	33	19	33	28	32	26	19	34	25	25
Last AA of Sig Pep	20	32	81	32	27	31	25	18	33	24	24
First AA of Sig Pep	1	1	1	1	1	1	1	1	-	1	1
AA SEQ ID NO:Y	1196	951	952	953	1197	954	955	956	156	856	1198
5' NT of First AA of Signal Pep	98	96	82	344	338	153	63	256	295	47	48
5' NT of Start Codon	98	96	82	344	338	153	63	256	295	47	48
3' NT of Clone Seq.	896	477	1925	2425	2460	1021	1155	727	1112	1238	1239
of Of Clone Seq.	∞	-	-		105		1	1	-	1	1
Total NT Seq.	896	477	1930	2425	2460	1021	1155	727	1112	1238	1239
× Si B Si ×	581	336	337	338	582	339	340	341	342	343	583
Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pCMVSport 3.0	pCMVSport 3.0
ATCC Deposit No:Z and Date	97974 04/04/97 209080 05/29/97	209126 06/19/97	PTA-622 09/02/99	PTA-1543 03/21/00	PTA-1543 03/21/00	209007 04/28/97 209083 05/29/97	209641 02/25/98	209603 01/29/98	209641 02/25/98	209463 11/14/97	209463 11/14/97
cDNA Clone ID	HSQE084	нѕоғр66	HSRFZ57	HSSGD52	HSSGD52	HSUBW09	HSVAT68	HSVBU91	HSXEC75	HSYBG37	HSYBG37
Gene No.	325	326	327	328	328	329	330	331	332	333	333

Last	AA of ORF	289	127	142	142	282	122	216	178	127	164	233	77
₹	of Secreted Portion	17	19	24	20	34	34	2	16	16	91	24	24
Last AA of	Sig Pep	16	18	23	19	33	33	1	15	15	15	23	23
First AA of	Sig Pep	-	1	1	-	1	1	1	1	1	1	1	1
~	DO:Y	959	1199	096	1200	961	1201	1202	962	1203	1204	963	1205
5' NT of First AA of		106	107	92	84	319	372	124	13	21	27	19	19
	Clone of Start Seq. Codon	106	107	92	84	319	372		13	21	27	19	19
3' NT of	Clone Seq.	1304	1333	1148	1140	1341	738	807	839	871	881	754	810
5' NT of	Clone Seq.	-	2	0	22	1	159	1	1		1	1	1
	Total NT	3cq.	1333	1147	1140	1341	738	935	6£8	871	881	754	810
NT SEQ	ДÖ,	344	584	345	585	346	586	587	347	588	589	348	290
		Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC	Deposit No:Z and Date	209124	209124	209124 06/19/97	209124 06/19/97	PTA-843 10/13/99	PTA-843 10/13/99	PTA-843 10/13/99	209877 05/18/98	209877 05/18/98	209877 05/18/98	209241 09/12/97	209241 09/12/97
	cDNA	HSZAF47	HSZAF47	HTADX17	HTADX17	HTAEE28	HTAEE28	HTAEE28	HTECC05	HTECC05	HTECC05	HTEDY42	HTEDY42
	Gene	334	334	335	335	336	336	336	337	337	337	338	338

Last AA of ORF	298	46	257	257	94	82	09	312	142	113	58	4
	2	,	2	2	<u>,</u>			(T)				
First AA of Secreted Portion	23	25	20	20	2	2	2	26	25	25	20	21
Last AA of Sig Pep	22	24	19	19	1	1	1	. 25	24	24	19	20
First AA of Sig Pep	-	1	1	1	1	1	1	1	1	1	-	-
AA SEQ ID NO:Y	964	965	996	1206	1207	1208	1209	<i>L</i> 96	896	1210	696	026
5' NT of First AA of Signal Pep	59	231	56	145	1	1081	029	121	188	187	22	164
3' NT of 5' NT Clone of Start Seq. Codon	59	231	26	145				121	188	187	22	164
	1022	1028	8/6	1092	133	937	908	1113	738	745	752	808
5' NT of Clone Seq.	20	1	-	-	-	754	-	-	1	-		-1
Total NT Seq.	1022	1028	876	1092	284	1494	1014	1113	738	745	752	808
× S B S ×	349	350	351	591	592	593	594	352	353	295	354	355
Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC Deposit No:Z and Date	97922 03/07/97 209070 05/22/97	209324 10/02/97	PTA-842 10/13/99	PTA-842 10/13/99	PTA-842 10/13/99	PTA-842 10/13/99	PTA-842 10/13/99	209568 01/06/98	209090 06/05/97	209090	209244 09/12/97	203648 02/09/99
cDNA Clone ID	нтеев42	HTEFU65	HTEGI42	HTEGI42	HTEGI42	HTEG142	HTEGI42	нтени31	нтени93	нтени93	HTEIP36	HTELP17
Gene No.	339	340	341	341	341	341	341	342	343	343	344	345

Last AA of	ORF	158	68	38	38	181	822	2	71	101	188	170	170	84
			<u></u>						6					4
First AA	Secreted Portion	18	28	16	16	23	2	21	29	44	18	19	19	34
Last AA of Sig	Pep	17	27	15	15	22	1	20	28	43	11	18	18	33
First AA of Sig	Рер	1	1	_	1	1	1	1	1	1	1	-1	1	1
AA SEQ ID	NO:Y	126	972	973	1211	974	975	1212	926	216	8/6	1213	1214	626
5' NT of First AA of Signal	Pep	15	365	231	224	527	30	335	33	73	124	189	110	110
5' NT of Start	Codon	15	365	231	224	527	30		33	73	124	189	110	110
	Seq.	1898	813	1818	2036	1569	2762	2694	926	818	2248	2214	928	978
5' NT of Clone	Seq.	1	1	1	П	198		21	1	,	-	1157	1	1
Total	NT Seq.	1898	813	1818	2036	1650	2762	2694	926	818	2248	2298	928	876
NT SEQ D	ÿ×	356	357	358	965	359	360	597	361	362	363	598	299	364
•	Vector	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pCMVSport 2.0	pCMVSport 2.0	pCMVSport 2.0	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC Deposit No:Z	and Date	PTA-1544 03/21/00	203570 01/11/99	PTA-844 10/13/99	PTA-844 10/13/99	209853 05/07/98	PTA-868 10/26/99	PTA-868 10/26/99	209241 09/12/97	209641 02/25/98	PTA-1543 03/21/00	PTA-1543 03/21/00	PTA-1543 03/21/00	PTA-2081 06/09/00
	cDNA Clone ID	HTELS08	HTEPG70	HTHCA18	HTHCA18	HTJMA95	HTJML75	HTJML75	HTLAA40	HTLEP53	HTLFE57	HTLFE57	HTLFE57	HTLIV19
	Gene No.	346	347	348	348	349	350	350	351	352	353	353	353	354

	-			F										
Last	AA of ORF	40	29	190	61	61	322	13	362	362	415	71	71	230
Fir	of Secreted Portion	27	24	25	21	21	2	10	23	25	23	30	30	25
Last AA of	Sig Pep	26	23	24	20	20	1	6	22	24	22	29	29	24
	Sig Pep	1	-	-	1	1	1	1	1	1	1	1	1	1
AA SEQ	HÖ.Y	980	981	982	983	1215	1216	1217	984	1218	1219	985	1220	986
5' NT of First AA of	Signal Pep	7	43	155	30	23	71	1555	178	302	92	2365	530	118
S' NT	Clone of Start Seq. Codon	7	43	155	30	23			178	302	92	2365	530	118
3' NT of	Clone Seq.	300	1019	946	1949	408	1274	1622	1374	1507	1404	3431	1598	1481
5' NT of	Clone Seq.	-	4	-	1	1	885	1	,1	118	1	2141	306	1
	Total NT Seq.	300	1019	946	1949	408	1299	1669	1374	1515	1404	3435	1598	1481
NT SEQ	A Š×	365	366	367	368	009	109	602	369	603	604	370	605	371
	Vector	pBluescript SK-	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
ATCC	Deposit No:Z and Date	209241 09/12/97	209244 09/12/97	203081 07/30/98	PTA-843 10/13/99	PTA-843 10/13/99	PTA-843 10/13/99	PTA-843 10/13/99	209511 12/03/97	209511 12/03/97	209511 12/03/97	209423 10/30/97	209423 10/30/97	PTA-871 10/26/99
	cDNA Clone ID	HTNBO91	HTODK73	HTOHD42	HTOHM15	HTOHM15	HTOHM15	HTOHM15	HTPBW79	HTPBW79	HTPBW79	HTPCS72	HTPCS72	нтрін83
	Gene No.	355	356	357	358	358	358	358	359	359	359	360	360	361

										,	,			
Last	AA of ORF	140	98	37	133	42	42	92	101	201	99	52	73	54
First AA	of Secreted Portion	25	2	35	23	18	18	2	22	22	19	23	18	18
Last AA of	Sig Pep	24	1	34	22	17	17	1	26	21	18	22	17	17
Ţ	Sig Pep	1	1	1	1	1	1	1	1	1	1	1	1	1
AA SEQ	NO:Y	1221	1222	987	988	986	1223	1224	066	991	992	993	994	1225
S' NT of First AA of	Signal Pep	111	96	170	133	95	100	175	334	217	328	72	1085	197
S' NT	of Start Codon	111		170	133	95	100		334	217	328	72	1085	197
3' NT of	Clone Seq.	530	1046	652	1111	2058	819	501	1963	1583	8682	1505	2135	1265
s. NT of	Clone Seq.	-	329	1	1	1	1	-	1	1	211	1	1	1
	Total NT Seq.	530	1046	652	1711	2058	819	501	1963	1583	2398	1505	2135	1265
NT SEQ	ВÄ×	909	607	372	373	374	809	609	375	376	377	378	379	610
	Vector	Uni-ZAP XR	Uni-ZAP XR	pBluescript	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pSport1	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	ZAP Express	ZAP Express
ATCC	Deposit No:Z and Date	PTA-871 10/26/99	PTA-871 10/26/99	209138 07/03/97	209641 02/25/98	PTA-841 10/13/99	PTA-841 10/13/99	PTA-841 10/13/99	209086 05/29/97	209746 04/07/98	209580 01/14/98	203648 02/09/99	209407 10/23/97	209407 10/23/97
	cDNA Clone ID	нтрін83	нтРІН83	HTSEW17	HTTBI76	HTTBS64	HTTBS64	HTTBS64	HTWCT03	HTXDW56	HTXJM03	HTXON32	HUDBZ89	HUDBZ89
	Gene No.	361	361	362	363	364	364	364	365	366	367	368	369	369

Last	AA of ORF	159	145	20	151	142	119	90\$	225	462	174	45	186	164
First AA	of Secreted Portion	27	27	30	21	23	23	20	20	31	24	21	35	35
Last AA of	Sig Pep	26	26	29	20	22	22	19	19	30	23	20	34	34
First AA of	Sig Pep	1	1	1	I	1	1	1	1	1	1	1	1	1
AA SEQ	ID NO:Y	995	1226	966	<i>L</i> 66	1227	1228	866	666	1229	1230	1000	1001	1231
5' NT of First AA of	Signal Pep	49	74	123	286	144	55	74	280	281	179	14	111	96
S' NT	of Start Codon	49	74	123	286	144	55	74	280	281	179	14	111	96
3. NT of	Clone Seq.	1193	1012	898	853	754	<i>L</i> 99	1757	2561	1997	1020	1502	1015	1006
5° NT of	Clone Seq.	-		1	-	-	1	99	-	1098	1	ī	-	1
	Total NT Seq.	1193	1012	898	853	754	<i>L</i> 99	1757	2561	2025	1020	1502	1015	1006
NT SEO	́ Bё×	380	611	381	382	612	613	383	384	614	615	385	386	919
	Vector	pSportl	pSportl	pSportl	Lambda ZAP II	Lambda ZAP II	Lambda ZAP II	Lambda ZAP II	pSport1	pSport1	pSport1	Uni-ZAP XR	pSport	pSport1
ATCC	Deposit No:Z and Date	PFA-1543 03/21/00	PTA-1543 03/21/00	209641 02/25/98	209568 01/06/98	209568 01/06/98	209568 01/06/98	209746 04/07/98	209651 03/04/98	209651 03/04/98	209651 03/04/98	209603 01/29/98	PTA-2076 06/09/00	PTA-2076 06/09/00
	cDNA Clone ID	HUFBY15	HUFBY15	HUFCJ30	HUKAH51	HUKAH51	HUKAHSI	HUKBT29	HUSXS50	HUSXS50	HUSXS50	HUVEB53	HVARW53	HVARW53
	Gene No.	370	370	371	372	372	372	373	374	374	374	375	376	376

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Last AA of	OK	168	53	169	48	43	75	187	187	105	105	<i>L</i> 9	61	52
	Secreted Portion	31	31	31	22	2	19	20	20	22	22	56	26	2
Last AA of Sig	Pep	30	30	30	21	1	18	19	61	21	21	25	25	1
First AA of Sig	Pep	1	1		1	1	1	1	1	1	1	1	1	1
AA SEQ ID	NO:Y	1002	1232	1233	1003	1004	1005	1006	1234	1007	1235	1008	1236	1009
5' NT of First AA of Signal	Pep	322	322	312	23	581	156	37	35	243	233	1342	132	271
	Codon	322	322	312 .	22	581	156	37	35	243	233	1342	132	271
3' NT of Clone	Seq.	3308	3306	2194	998	1769	1051	1317	1315	1138	1138	1841	314	1677
	Seq.	1	1	1	1	625	1	3	1	1	1	1	-	1
Total	NT Seq.	3308	3306	2194	998	1769	1021	1317	1315	1138	1138	1841	314	1677
SEQ EQ	XO:	387	219	618	388	389	330	391	619	392	620	393	621	394
	Vector	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0	pCMVSport 3.0
ATCC Deposit No:Z	and Date	203570 01/11/99	203 <i>57</i> 0 01/11/99	203570 01/11/99	209463 11/14/97	PTA-1543 03/21/00	PTA-867 10/26/99	PTA-499 08/11/99	PTA-499 08/11/99	209641 02/25/98	209641 02/25/98	209641 02/25/98	209641 02/25/98	PTA-1543 03/21/00
	cDNA Clone ID	HWAAD63	HWAAD63	HWAAD63	HWABA81	HWADJ89	HWBAR88	HWBCB89	HWBCB89	HWBCP79	HWBCP79	HWBDP28	HWBDP28	HWBFX31
	Gene No.	377	377	377	378	379	380	381	381	382	382	383	383	384

		J	r+	\neg			1			T.		T		٦		T						
	Last	AA of	ORF		188	102		188	742	1	CO		62		46		4	310		310		310
	First AA	oę	Secreted	Portion	31	32		31	27	9	61		32		27			31		31		31
Last	AA of AA of	Sig	Pep	-	30	31		30	26	,	×		31		. 56		22	30		30		30
First	AA of	Sig	Pep					_	-	ŀ	-		1		-		-	1		1		1
ΑA		<u>a</u>	NO:Y		1010	1237		1238	1011	0,0	1012		1013		1014		1015	1016		1239		1240
S' NT of First	AA of	Signal	Pep		131	209		101	169	9	129		190		157		319	468		468		468
	5' NT	Clone of Start Signal	Codon		131	500		101	169	١	129		190		157		319	468		468		468
3. NT	of	Clone	Seq.		1529	1796		2136	3282	ٳ	831		3337		1440		838	4386		4385		4386
5' NT	of	Clone	Seq.		95				1	,	~		_		-		_	П		1		1
		Total	Ľ	Seq.	1529	1796		2136	3282		831		3337		1440		838	4386		4385		4386
Ä	SEQ	白	ÖN.	×	395	622		623	396		397		398		399		94	401		624		625
				Vector	pCMVSport 3.0	pCMVSport	3.0	pCMVSport	pCMVSport	0.0	pSportl		pCMVSport	3.0	Uni-ZAP XR		Uni-ZAP XR	Uni-ZAP XR		Uni-ZAP XR		Uni-ZAP XR
	ATCC	Deposit No:Z	and Date		203181	203181	86/60/60	203181	203027	00/07/00	203081	01/130/198	203517	12/10/98	209580	01/14/98	203570	PTA-1543	03/21/00	PTA-1543	03/21/00	PTA-1543 03/21/00
			cDNA	Clone ID	HWHHL34	HWHHL34		HWHHL34	НЖНОЅЅЅ		HWLIH65		HYAAJ71		HYBAR01		HYBBE75	HAPSA79		HAPSA79		HAPSA79
			Gene	Š.	385	385		385	386		387		388		389		390	391		391		391

Table 1B (Comprised of Tables 1B.1 and 1B.2)

The first column in Table 1B.1 and Table 1B.2 provides the gene number in the application corresponding to the clone identifier. The second column in Table 1B.1 and Table 1B.2 provides a unique "Clone ID:" for the cDNA clone related to each contig sequence disclosed in Table 1B.1 and Table 1B.2. This clone ID references the cDNA clone which contains at least the 5' most sequence of the assembled contig and at least a portion of SEQ ID NO:X as determined by directly sequencing the referenced clone. The referenced clone may have more sequence than described in the sequence listing or the clone may have less. In the vast majority of cases, however, the clone is believed to encode a full-length polypeptide. In the case where a clone is not full-length, a full-length cDNA can be obtained by methods described elsewhere herein. The third column in Table 1B.1 and Table 1B.2 provides a unique "Contig ID" identification for each contig sequence. The fourth column in Table 1B.1 and Table 1B.2 provides the "SEQ ID NO:" identifier for each of the contig polynucleotide sequences disclosed in Table 1B.

Table 1B.1

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The fifth column in Table 1B.1, "ORF (From-To)", provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence "SEQ ID NO:X" that delineate the preferred open reading frame (ORF) shown in the sequence listing and referenced in Table 1B.1, column 6, as SEQ ID NO:Y. Where the nucleotide position number "To" is lower than the nucleotide position number "From", the preferred ORF is the reverse complement of the referenced polynucleotide sequence. The sixth column in Table 1B.1 provides the corresponding SEQ ID NO:Y for the polypeptide sequence encoded by the preferred ORF delineated in column 5. In one embodiment, the invention provides an amino acid sequence comprising, or alternatively consisting of, a polypeptide encoded by the portion of SEQ ID NO:X delineated by "ORF (From-To)". Also provided are polynucleotides encoding such amino acid sequences and the complementary strand thereto. Column 7 in Table 1B.1 lists residues comprising epitopes contained in the polypeptides encoded by the preferred ORF (SEQ ID NO:Y), as predicted using the algorithm of Jameson and Wolf, (1988) Comp. Appl. Biosci. 4:181-186. The Jameson-Wolf antigenic analysis was performed using the computer program PROTEAN (Version 3.11 for the Power MacIntosh, DNASTAR, Inc., 1228 South Park Street Madison, WI). In specific embodiments, polypeptides of the invention comprise, or alternatively consist of, at least one, two, three, four, five or more of the predicted epitopes as described in Table 1B. It will be appreciated that depending on the analytical criteria used to predict antigenic determinants, the exact address of the determinant may vary slightly.

Column 8 in Table 1B.1 provides a chromosomal map location for certain polynucleotides of the invention. Chromosomal location was determined by finding exact matches

to EST and cDNA sequences contained in the NCBI (National Center for Biotechnology Information) UniGene database. Each sequence in the UniGene database is assigned to a "cluster"; all of the ESTs, cDNAs, and STSs in a cluster are believed to be derived from a single gene. Chromosomal mapping data is often available for one or more sequence(s) in a UniGene cluster; this data (if consistent) is then applied to the cluster as a whole. Thus, it is possible to infer the chromosomal location of a new polynucleotide sequence by determining its identity with a mapped UniGene cluster.

A modified version of the computer program BLASTN (Altshul, et al., J. Mol. Biol. 215:403-410 (1990), and Gish, and States, Nat. Genet. 3:266-272) (1993) was used to search the UniGene database for EST or cDNA sequences that contain exact or near-exact matches to a polynucleotide sequence of the invention (the 'Query'). A sequence from the UniGene database (the 'Subject') was said to be an exact match if it contained a segment of 50 nucleotides in length such that 48 of those nucleotides were in the same order as found in the Query sequence. If all of the matches that met this criteria were in the same UniGene cluster, and mapping data was available for this cluster, it is indicated in Table 1B under the heading "Cytologic Band". Where a cluster had been further localized to a distinct cytologic band, that band is disclosed; where no banding information was available, but the gene had been localized to a single chromosome, the chromosome is disclosed.

Once a presumptive chromosomal location was determined for a polynucleotide of the invention, an associated disease locus was identified by comparison with a database of diseases which have been experimentally associated with genetic loci. The database used was the Morbid Map, derived from OMIMTM and National Center for Biotechnology Information, National Library of Medicine (Bethesda, MD) 2000;. If the putative chromosomal location of a polynucleotide of the invention (Query sequence) was associated with a disease in the Morbid Map database, an OMIM reference identification number was noted in column 9, Table 1B.1, labelled "OMIM Disease Reference(s). Table 5 is a key to the OMIM reference identification numbers (column 1), and provides a description of the associated disease in Column 2.

Table 1B.2

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Column 5, in Table 1B.2, provides an expression profile and library code:count for each of the contig sequences (SEQ ID NO:X) disclosed in Table 1B, which can routinely be combined with the information provided in Table 4 and used to determine the tissues, cells, and/or cell line libraries which predominantly express the polynucleotides of the invention. The first number in Table 1B.2, column 5 (preceding the colon), represents the tissue/cell source identifier code corresponding to the code and description provided in Table 4. The second number in column 5 (following the colon) represents the number of times a sequence corresponding to the reference

polynucleotide sequence was identified in the corresponding tissue/cell source. Those tissue/cell source identifier codes in which the first two letters are "AR" designate information generated using DNA array technology. Utilizing this technology, cDNAs were amplified by PCR and then transferred, in duplicate, onto the array. Gene expression was assayed through hybridization of first strand cDNA probes to the DNA array. cDNA probes were generated from total RNA extracted from a variety of different tissues and cell lines. Probe synthesis was performed in the presence of ³³P dCTP, using oligo (dT) to prime reverse transcription. After hybridization, high stringency washing conditions were employed to remove non-specific hybrids from the array. The remaining signal, emanating from each gene target, was measured using a Phosphorimager. Gene expression was reported as Phosphor Stimulating Luminescence (PSL) which reflects the level of phosphor signal generated from the probe hybridized to each of the gene targets represented on the array. A local background signal subtraction was performed before the total signal generated from each array was used to normalize gene expression between the different hybridizations. The value presented after "[array code]:" represents the mean of the duplicate values, following background subtraction and probe normalization. One of skill in the art could routinely use this information to identify normal and/or diseased tissue(s) which show a predominant expression pattern of the corresponding polynucleotide of the invention or to identify polynucleotides which show predominant and/or specific tissue and/or cell expression.

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TABLE 1B.1

41		_			AA			
ö	cDNA Clone	Contig ID:	SEQ ID NO:	ORF (From-To)	SEQ ID NO. V	Predicted Epitopes	Cytologic Band	OMIM Disease Reference(s):
-	117001100	004124	4	157 777	707	Dra 67 to Ash 67	11014-013	105210 106210 106210 106210
_	HZCDU03	1004124	11	/// - /CT	070	110-02 to Asp-07,	CId-LIdit	102/12, 100210, 100210, 100210, 100210,
						Arg-74 to Gly-80,		10/2/1, 114550, 115500, 136550, 151590,
						Gln-146 to Glu-168.		179615, 179615, 179616, 180385, 194070,
				-				194070, 194070, 245349, 602092
	H2CBU83	745366	402	157 - 312	1017			
2	H2MAC30	544957	12	157 - 375	627	Pro-54 to Gly-67.		
3	H6EAB28	1352227	13	115 - 414	628	Ser-39 to Gly-46,	7p22	600259, 600259
						Leu-49 to Ala-62,		
						Lys-79 to Ala-93,		
						Gly-95 to Thr-100.		
	H6EAB28	589947	403	116 - 346	1018	Ala-29 to Thr-37,		
						Pro-39 to Leu-63.		
4	H6EDC19	543259	14	389 - 733	629	Arg-5 to Pro-12.		
5	H6EDF66	520498	15	146 - 538	930			
9	H6EDX46	1352262	16	229 - 774	631	Arg-21 to Leu-26,	12q15	181430, 600698, 600698, 600698, 600698,
						Arg-88 to Asn-104,		600808, 602116
						Arg-111 to Ser-116,		
,						Arg-154 to Lys-160,		
						Cys-164 to Asp-169.		
	H6EDX46	637786	404	128 - 382	1019	Arg-21 to Leu-26.		
7	HACBD91	637482	17	117 - 266	632		3q13.33	600882
∞	HACCI17	891114	18	461 - 1114	633	Ser-201 to Tyr-217.	22q11.21	123620, 151410, 600850
	HACCI17	731877	405	135 - 353	1020			
6	HAGAI85	381942	19	166 - 255	634	Ser-24 to Trp-30.	9q31-q32	109400, 132800, 132800, 154400, 186855, 223900, 253800, 253800, 278700, 602088
9	HAGAO26	561996	20	251 - 439	635		7933	180105, 222800

Cytologic OMIM	×	4p16.3 134934, 134934, 134934, 134934, 134934, 134934, 134934, 134900, 180072, 180072, 194190, 252800, 252800, 600965			5q31.3 131400, 159000, 180071, 181460, 272750, 600807, 601596, 602089			5q31.3 131400, 159000, 180071, 181460, 272750,	600807, 601596, 602089		179095, 181460, 192974, 192974, 600807, 601596, 601692, 601692, 601692, 601692, 602089, 602121, 602460		5q12-q13 126060, 143200, 143200, 181510, 253200, 268800, 268800, 600354, 600354, 600354,	
Predicted Epitopes		Ser-36 to Gly-41, Pro-43 to Ser-49.	Leu-31 to Phe-38, Glu-47 to Trp-52.	Leu-31 to Phe-38, Glu-47 to Trp-52.				Pro-70 to Arg-77,	Tyr-102 to Thr-107.		Gln-81 to Lys-88, Asp-93 to Lys-102, Asn-107 to Leu-116, Met-129 to Glu-141, Glu-150 to Asp-157, Lys-176 to Glu-185,	Glu-333 to 1yr-349, Cys-393 to Leu-403, Gln-423 to Gly-429.	Pro-186 to Tyr-196, Leu-294 to Leu-300,	
AA SEQ	NO: Y	636	637	1021	638	1022	639	640		1023	5		642	
ORF	X (Trought)	318 - 596	45 - 410	52 - 405	251 - 844	128 - 262	325 - 525	311 - 1261		1 - 54			109 - 1797	
SEQ	×	21	22	406	23	407	24	25		408			27	
Contig		597444	1352199	543617	778820	381964	490848	727543		371337			1352364	
cDNA Clone		HAGD135	HAGDS35	HAGDS35	HAGFY16	HAGFY16	HAIB071	HAIBP89	_	HAIBP89			HAJAN23	
Gene No:	 	11	12		13		14	15.					17	

OMIM Disease Reference(s):				157640, 174900, 236730, 600512					152200, 167000, 600320, 600883, 602544						180105, 222800, 274180													
Cytologic Band				10q23.33					6q27						7q34													
Predicted Epitopes	Phe-513 to Ser-522.			Gly-19 to Ser-27,	Gln-39 to Gly-45,	Gln-48 to Ala-55,	Ala-75 to Thr-80,	Thr-198 to Gly-211.	Asp-31 to Pro-36,	Ser-88 to Gln-95,	Ala-163 to Glu-171.	Asp-31 to Pro-36,	Ser-88 to Gln-95.		Leu-8 to Thr-16,	Gly-93 to Ala-105,	Arg-136 to Thr-142,	Lys-195 to Gln-200,	Lys-241 to His-247,	Gly-255 to Gln-270,	Gln-288 to Leu-293,	Thr-316 to Asp-328,	Gly-348 to Pro-355,	Asp-408 to Met-415.	Ser-39 to Asn-47.	Met-1 to Ser-6.	Ala-27 to Asp-34,	Tyr-116 to Leu-125.
AA SEQ ID NO: Y		1024	643	644					645			1025		1026	646										1027	647	648	
SEQ ORF ID NO: (From-To)	1	120 - 629	262 - 423	49 - 1872					136 - 711			115 - 651		323 - 349	1495 - 2757										226 - 369	279 - 518	85 - 853	
SEQ ID NO: X		409	28	29					30			410		411	31										412	32	33	
Contig ID:		872551	638516	618530					904749			900286		618906	905695										823350	647105	892971	
Gene CDNA Clone No: ID		HAJAN23	HAJBR69	HAJBZ75					HAMFC93			HAMFC93		HAMFC93	HAMFE15										HAMFE15	HAMFK58	HAMGR28	
Gene No:			18	19					20						21											22	23	

OMIM Disease Reference(s):																									
Cytologic Band																		-							
Predicted Epitopes	Ala-27 to Asp-34, Tyr-116 to Leu-125, Arg-185 to Cys-194.		Glu-61 to Gln-66, A1a-93 to Glu-98	Pro-31 to Thr-48	Arg-62 to Gly-70,	Ala-74 to Glu-87,	Lys-123 to Asp-129,	Pro-162 to Gly-167,	Glu-170 to Gly-189,	Arg-220 to Asn-228.	Pro-28 to Thr-45,	Arg-59 to Gly-67,	Ala-71 to Glu-84,	Lys-120 to Asp-126,	Pro-159 to Gly-164,	Glu-167 to Gly-186,	Arg-217 to Asn-225,	Glu-245 to Ala-255,	Gly-282 to Gly-297,	Pro-312 to Gly-324,	Thr-356 to Lys-364,	Gly-366 to Thr-372,	Lys-377 to Ala-383,	Gly-397 to Thr-407,	Gln-23 to Asp-30,
AA SEQ ID NO: Y	1028	649	1029	1030	2						1031														650
SEQ ORF ID NO: (From-To)	40 - 651	520 - 675	125 - 418	70-1245	CF31 : 0/		_				78 - 1379											,			251 - 817
SEQ ID NO: X	413	34	414	415	CTŁ						416														35
Contig ID:	748223	845690	852533	844216	01710						692291														769555
Gene cDNA Clone	HAMGR28	HANGG89	HANGG89	HANGG80	1000000						HANGG89														HAPOM49
Gene No:		24																			_				25

OMIM Disease Reference(s):			٠																									
Cytologic Band																					-							
Predicted Epitopes	Lys-66 to Cys-87.	Met-1 to Cys-21,	Cys-41 to Asp-59,	Pro-104 to His-116.	Glu-42 to Pro-53,	Ser-67 to Tyr-79,	Phe-137 to Leu-143,	Ser-180 to Arg-186,	Trp-188 to Gly-195,	Pro-210 to Arg-216,	Thr-222 to Asp-243.	Glu-42 to Pro-53,	Ser-67 to Thr-73,	Ala-84 to Leu-90.		Lys-25 to Ser-36,	Ser-53 to Glu-60,	Thr-70 to Arg-75,	Arg-111 to Thr-119,	Lys-204 to Leu-248.	Lys-25 to Ser-36,	Ser-53 to Glu-60,	Thr-70 to Arg-75,	Arg-111 to Thr-119,	Glu-161 to Leu-189.	Ile-25 to Trp-30.	Ser-35 to Ser-44,	Ser-80 to Leu-91,
AA SEQ ID	1	1032			651							1033			652	653					1034					654	559	
SEQ ORF D NO: (From-To)		448 - 816			59 - 850							54 - 329			385 - 807	97 - 840					899 - 66					252 - 446	143 - 1300	
SEQ ID NO:	4	417			36							418			37	38					419					39	40	
Contig ID:		722386			1352278							684272			834358	1352276					667830					635514	845965	
cDNA Clone ID		HAPOM49			HAPPW30							HAPPW30			HAPUC89	HATAC53					HATAC53					HATBR65	HATDF29	
Gene No:					56								_		27	28									_	50	30	

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	cDNA Clone Config	NO. A	SEQ OKF ID NO: (From-To) X	SEQ NO: Y	Fredicted Epitopes	Cytologic	Disease Reference(s):
1			 		Asp-143 to Leu-150,		
					Lys-166 to Ser-171,		
					Ser-208 to Gly-213,		
					Lys-239 to Leu-244, Glu-317 to Asn-324.		
HATEE46	565618	41	241 - 402	959			
ĺ	639009	42	253 - 399	657	Asn-34 to Lys-42.	61	
HAUA183	383592	420	575 - 643	1035	Ala-17 to Lys-23.		
HBAF133	625916	43	60 - 392	859	Gln-66 to Cys-71,	14q32	123270, 245200, 251600, 270100, 276900
					Thr-76 to Gly-81,		
					His-87 to Asp-92.	٠	
HBAFV19	843036	44	6-11-9	659	Pro-12 to Phe-18,		
					Ser-139 to Pro-146,		
					Asp-162 to Arg-173,		
					Thr-188 to Glu-204, Lys-245 to Gly-258.		
HBAMB15	671835	45	390 - 569	099		2p16	126600, 126600, 136435, 160980, 600678
HBCPB32	1352403	46	88 - 693	199		4	
HBCPB32	1045580	421	629 - 68	1036			
HBCQL32	1134954		26 - 268	662		17	
HBCQL32	1027748	422	760 - 1002	1037			
HBGBA69	1352289	48	124 - 843	699	Pro-51 to Asp-56,		
					Gly-95 to Thr-105,		
					Val-132 to Ala-138,		
					Pro-229 to Leu-240.		
HBGBA69	709658	423	62 - 244	1038	Thr-52 to Gly-57.		
HBIAE26	514418	49	75 - 194	664	Ser-22 to Lys-27.		
HBIMB51	963208	50	98 - 535	999	His-24 to Ala-29,		

Fredicted Epitopes
Glu-42 to Glu-49,
Arg-63 to Thr-80,
Gln-100 to Lys-119,
Lys-141 to Gln-146.
His-24 to Ala-29,
Glu-42 to Glu-49.
Gly-32 to Gly-37,
Glu-78 to His-87,
Tyr-102 to Ala-107,
Pro-115 to Val-122,
yr-1′
Gly-32 to Gly-37,
Glu-78 to His-87,
Tyr-102 to Ala-107,
Pro-115 to Val-122,
Lys-164 to Gln-171
Gly-32 to Gly-37,
Glu-78 to His-87,
Tyr-102 to Ala-107,
Pro-115 to Val-122
Lys-82 to Pro-87,
Leu-110 to Lys-129.
6-0
Gln-23 to Asn-31,
Tyr-42 to Ser-58.
Pro-29 to Gly-46,
Lys-48 to Gly-55,
Lys-67 to Gly-80,

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Mino	Disease	Reference(s):																			And the second s		106180, 109270, 109270, 109270, 109270,	109270, 120150, 120150, 120150, 138700, 139250-148065-148080-150200-154275	171190, 176960, 185800, 221820, 249000.	253250, 600525, 600852, 601844		
	Cytologic						-				-										1	-	17q22-q23					
3	Predicted Epitopes		Lys-100 to Pro-115,	Arg-121 to Gly-127,	Asn-139 to Gly-149,	Ser-179 to Arg-185,	Asp-191 to Gly-196,	Lys-219 to Gly-224.	Pro-29 to Gly-46,	Lys-48 to Gly-55,	Lys-67 to Gly-80,	Gly-89 to Asn-99.	Pro-29 to Gly-46,	Lys-48 to Gly-55,	Lys-67 to Gly-80,	Lys-100 to Pro-115,	Arg-121 to Gly-127,	Asn-139 to Gly-149,	Ser-179 to Arg-185,	Asp-191 to Gly-196, 1 ys-219 to Gly-224.	Arg-37 to Gly-42.		Arg-36 to Pro-43.			:		
AA	3 5	NO: Y							1043				1044								0/9	671	672				673	674
	OKF (From-To)	X							66 - 365	•			64 - 801								156 - 407	77 - 262	560 - 733				137 - 388	168 - 338
	SEC IN NO	×							428				429								55	56	57				58	59
	Contig	•							899397				902207								668268	526797	842802				625923	544988
	cDNA Clone	}							HBJNC59				HBJNC59								HBMCI50	HBNAW17	HBXFL29				HCACU58	HCDAF84
	Gene	•																			45	46	47				48	49

																								Γ			
	OMIM Disease Reference(s):	103850, 114835, 116800, 140100, 140100, 192090, 192090, 192090, 192090, 192090, 245900,	245900, 276600, 600223				120070, 120131, 120131, 138030, 259900									601362							and the latest	176261. 601399			
	Cytologic Band	16q22.1					2q36.1									10p13								21022.2		22q13.33	•
	Predicted Epitopes	His-44 to Pro-50, Glu-90 to Glu-96,	Gln-111 to Glu-117,	Ser-143 to Gly-151,	Pro-199 to Ala-216,	Gly-264 to Asp-272.	Lys-50 to Asp-66,	Pro-68 to Glu-77,	Glu-102 to Glu-107,	Glu-131 to Leu-146,	Ala-175 to Glu-183,	Phe-205 to Lys-216,	Val-263 to Thr-281,	Pro-304 to Ala-313.	Lys-50 to Leu-69.	Asn-23 to Ser-32,	Trp-61 to Ser-68,	Ala-130 to Ala-135,	Thr-141 to Gly-148,	Asn-176 to Gly-182,	Pro-197 to Glu-205,	His-211 to Glu-222,	Gln-242 to Ile-248,	Asn-28 to Pro-34.	Gln-189 to Gly-195.	Met-1 to Ala-8,	Ser-51 to Leu-62,
AA	SEQ ID	675					9/9								1045	677								87.9	629	089)))
	SEQ ORF ID NO: (From-To)	166 - 1125					165 - 1175								165 - 482	113 - 931								134 - 316	188 - 862	12 - 281	1
	SEQ ID NO:	09					19								430	62								63	64	65	}
	Contig ID:	634016					728432								494346	612796								634967	748245	1143407	4
	Gene cDNA Clone No: ID	HCE2F54					HCE3G69								HCE3G69	HCE5F43								HCFFA88	HCEFB69	HCEFB80	
	Gene No:	20					51									52								53	24	55	}

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cDNA Clone	5.0	SEQ	ORF	SEQ	Predicted Epitopes	Cytologic	OMIM
		ED NO:	ID NO: (From-To)	NO: Y		Band	Disease Reference(s):
					Pro-70 to Lys-78.		
HCEFB80	1046853	431	5 - 274	1046	Met-1 to Ala-8.		
HCEGR33	425212	99	243 - 338	681			
\vdash	941941	19	117 - 437	682	Gly-36 to Thr-41,	1921.3	104770, 107670, 110700, 145001, 146760,
					Pro-99 to Cys-106.		146790, 191315, 601412, 601652, 601863, 602491
HCEWE17	893535	432	500 - 583	1047			
Т	460407	433	156 - 317	1048	His-12 to Lys-18,		
					Ala-20 to Ala-26,	-	
					Arg-30 to Trp-52.		
HCEWE20	543370	89	166 - 321	683	Ser-17 to Gln-22.		
HCGMD59	636078	69	438 - 662	684			
HCMSQ56	740781	0/	148 - 414	989	Pro-61 to Asp-68.	5q31	121050, 131400, 138040, 153455, 159000,
							179095, 181460, 192974, 192974, 600807,
_							601596, 601692, 601692, 601692, 601692, 602089, 602121, 602460
HCNDR47	1016919	71	21 - 401	989	Pro-71 to His-92.	1	
HCNDR47	863677	434	124 - 507	1049	Pro-71 to His-92.		
HCNDR47	874128	435	603 - 632	1050	Leu-1 to Thr-9.	•	
M70	637547	72	107 - 751	L89	Met-1 to Ser-6.	11q24	600359, 602574, 602574
HCNSM70	589445	436	161 - 436	1021	Met-1 to Ser-6.		
HCOOS80	1134974	22	36 - 512	889	Pro-39 to Leu-44,	17p13.2	
					Gln-80 to Pro-93, Pro-153 to Pro-158.		
HCOOS80	1045182	437	40 - 516	1052	Pro-39 to Leu-44,		
					Gln-80 to Pro-93, Pro-153 to Pro-158.		
HCOOS80	1045183	438	1 - 318	1053	Pro-12 to His-25.		

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	OMIM	Discuse Reference(s):																								133701, 168500, 171650, 176930, 176930, 600623, 600811, 600958		
	Cytologic	Dana						-																		11p11.2	119	
	Predicted Epitopes				Pro-54 to Phe-63,	Gly-115 to Gln-121,	Gln-136 to Ala-141,	Gln-164 to Leu-178,	Glu-194 to Trp-203,	Glu-215 to Arg-222,	Glu-296 to Gly-304.	Pro-54 to Phe-63,	Gly-115 to Gln-121,	Gln-136 to Ala-141,	Gln-164 to Leu-178,	Glu-194 to Trp-203,	Glu-215 to Asp-223.		Lys-28 to Thr-34.		Lys-21 to Gln-32,	Asp-117 to Glu-124,	Tyr-179 to Gly-184,	Asn-211 to Gly-217,	Leu-239 to Lys-264.	Asp-48 to Ser-54.	Ala-145 to Ser-154,	Ala-258 to Tyr-263, Ala-287 to Arg-297,
AA	SEQ	NO: Y	689	069	691							1054						1055	692	693	694				i	695	969	
	ORF	O: (From-10)	557 - 700	19-318	148 - 1176							247 - 978						155 - 886	37 - 159	138 - 335	118 - 942		•			568 - 894	199 - 1440	
	SEQ.	S X	74	75	9/							439						440	77	78	79					08	81	
	Contig		550208		1352416							1115089						889268	553621	628256	598019					499233	902513	
	cDN,	3	HCUIM65	HCWDS72	HCWEB58							HCWEB58						HCWEB58	HCWKC15	HCWLD74	HCYBG92					нрнев60	HDHMA45	
	Gene	; ,	8	65	99														29	89	69					70	71	

OMIM	Disease Reference(s):										-																		
Cytologic	Band					5914.3	4	_																					
Predicted Epitopes		Thr-306 to Met-316.	Ala-145 to Ser-154,	Ala-258 to Tyr-263,	Ala-287 to Arg-297, Thr-306 to Met-316	Gln-33 to Trp-49.	Giv-161 to Giv-172.	Ile-207 to Arg-212,	Asn-414 to Val-419,	Val-423 to Gln-428,	Val-436 to Gly-441,	Lys-467 to Leu-478,	Phe-497 to Ser-508,	Met-550 to Gly-560,	Glu-688 to Thr-697,	Ile-711 to Gly-720,	Ala-747 to Gly-759,	Leu-785 to Phe-791,	Ser-795 to Gln-800,	Thr-808 to Lys-813,	Ser-821 to Phe-832,	Thr-879 to Glu-889,	Leu-898 to Gln-904,	Gln-934 to Met-941.	Gln-33 to Trp-49,	Gly-161 to Gly-172,	Ile-207 to Arg-212,	Asn-414 to Val-419,	Val-423 to Gln-428,
AA SEQ	NO: Y		1056			697																			1057				
ORF	O: (From-To)		204 - 1445			259 - 3084																			69 - 2894				
SEQ	ID NO: X		441			82	}																		442				
Contig	Ä		812764			1062783																-			866429				
Gene cDNA Clone	Q		HDHIMA45			HDPBA28															•				HDPBA28				
Gene	ë					72	!																						

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Gene	Gene cuna Clone	Contig	2H2	OKF	SEC ES	Predicted Epitopes	Cytologic	Discoss	
ë	3	ä		ID NO: (From-10)	NO: Y		Dalid	Reference(s):	
						Val-436 to Gly-441,			
						Lys-467 to Leu-478,			_
						Phe-497 to Ser-508,			
						Met-550 to Gly-560,			
						Glu-688 to Thr-697,			
						Ile-711 to Gly-720,			
						Ala-747 to Gly-759,			
						Leu-785 to Phe-791,			
	-					Ser-795 to Gln-800.			٦
73	HDPCL63	1019008	83	35 - 835	869	Ile-4 to Glu-10,			_
						Gly-58 to Asp-64.			7
	HDPCL63	847045	443	260 - 733	1058	Lys-72 to Cys-80,			
						Leu-90 to Pro-96,	•		
						Ala-110 to Thr-119,			_
						Glu-121 to Gly-128,			
						Ser-140 to Lys-147.			
	HDPCL63	897484	444	605 - 961	1059	Pro-8 to Gln-13,			
				_		Thr-38 to Pro-46,			
						Pro-100 to Met-108, Pro-113 to Pro-118.			
74	HDPC025	460682	84	182 - 343	669	Pro-22 to His-33,			Γ
						Ser-42 to Trp-48.			
75	HDPCY37	837699	88	76 - 1809	700	Pro-23 to His-34,	12q13.3	181430, 232800, 600808, 601284, 601769,	
						Thr-64 to Trp-71.		601769, 602116	٦
	HDPCY37	604114	445	76 - 870	1060	Pro-23 to His-34,			
						Thr-64 to Trp-71,			
76	HDPFR02	808208	86	173.631	701	Gln-77 to Glv-77			Τ
	11011 002	07070		1/3 - 6/1	10/	014-12 (0 01)-17,			7

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S S	CDINA CIONE	Contrig	ID NO:	(From-To)		r reuticeu Epitopes	Band	Disease
	}	i	X	X	NO: Y			Reference(s):
						Arg-115 to Arg-125, His-138 to Pro-146.		
	HDPFB02	1056541	446	139 - 1086	1901	Met-1 to Gly-6,		
						Glu-81 to Gly-86,	·-	
						Glu-150 to Asp-159,		
						Ser-166 to Glu-173,		
						Ser-277 to Glu-291,		
						Leu-302 to Gly-312.		
	HDPFB02	997408	447	218 - 1123	1062	Arg-17 to Glu-24,		
	_	-				Glu-41 to Asp-46,		
					•	Val-76 to Arg-83,		
						Thr-104 to Gln-109.		
77	HDPFF39	588697	87	175 - 765	702	Ser-128 to Thr-133,	19q13.2-	107741, 113900, 122720, 122720, 126340,
						Thr-158 to Thr-166,	q13.3	126391, 130410, 134790, 138570, 160900,
						Leu-168 to Gly-175,		164731, 173850, 207750, 248600, 258501,
_						Ala-179 to Asp-196.		600040, 602225, 602225
78	HDPFP29	628254	88	293 - 451	703			
79	HDPGT01	771583	68	8 - 271	704	Cys-65 to Ser-71.	16q22.1	103850, 114835, 116800, 140100, 140100,
								192090, 192090, 192090, 192090, 245900, 245900, 276600, 600223
8	HDPHI51	460679	06	245 - 367	705	Gly-2 to Glu-7,		
					:	Arg-27 to Gly-34.		
81	HDPJF37	704487	16	196 - 369	902	Pro-27 to Gly-34.		
82	нDРЈМ30	879325	65	59 - 1633	<i>1</i> 0 <i>1</i>	Arg-15 to Val-22.	21q22.3	120220, 120240, 123580, 151385, 171860, 190685, 236100, 236200, 240300, 267750,
								000003, 0010/2, 001143
	HDPJM30	603517	448	259 - 438	1063	Pro-41 to Ala-55.		
83	HDPMM88	972734	93	100 - 2913	208	Met-1 to Ser-13,		

	OMIM	Reference(s):																											8p21.2-p21.1 138300, 240400, 602629	
	Cytologic Rand	Dallu													-														8p21.2-p21.1	
	Predicted Epitopes		Ser-45 to Phe-51,	Asn-103 to Lys-113,	Phe-135 to Gly-140,	Asp-165 to Pro-178,	Ser-224 to Ala-229,	Asn-283 to Arg-288,	Asp-347 to Tyr-352,	Thr-367 to Glu-372,	Gly-420 to Thr-425,	Glu-456 to Lys-462,	Phe-466 to Asn-474,	Glu-480 to Leu-485,	Asp-673 to Asp-681,	Gln-684 to Gly-689,	Leu-841 to Gly-874,	Gly-890 to Pro-900,	Ser-902 to Ser-911,	Leu-918 to Asp-924,	Ser-930 to Val-935.	Ser-28 to Phe-34,	Asn-86 to Tyr-93.					Ser-26 to Thr-31.	Ala-88 to Gln-98.	Met-1 to Ser-8.
AA	SEQ	NO: Y																				1064		1065	1066	1067	1068	1069	602	710
	ORF	X (From-10)																				141 - 467		44 - 181	419 - 439	111 - 146	167 - 334	28 - 186	118 - 573	252 - 980
	SEQ	S X																				449		450	451	452	453	424	94	95
	Contig	ï																				906121		902299	885059	874074	854246	854245	897276	683371
	CDN	3																				HDPMM88		HDPMM88	HDPMM88	HDPMM88	HDPMM88	HDPMM88	HDPOE32	Н DРОН06
	d)	ë Ž																											84	85

	OMIM	Reference(s):	222900													•														
	Cytologic	Danu	3q25.33			. 10																-								
	Predicted Epitopes		Lys-30 to Thr-35.			Lys-23 to Lys-31,	Ala-38 to Ser-43.			Lys-57 to Gly-64.	Met-1 to Trp-6,	Leu-22 to Thr-27,	Pro-44 to Thr-63.	Met-1 to Trp-6,	Leu-22 to Thr-27,	Pro-44 to Gly-58,	Ala-61 to Glu-74,	Pro-99 to Gly-111,	Cys-121 to Ser-127.	Met-1 to Trp-6,	Leu-22 to Thr-27.	Gln-75 to Cys-80,	Glu-97 to Lys-104,	Glu-114 to Ala-119,	Thr-177 to Gln-190,	Asn-230 to Trp-240,	Glu-269 to Arg-274,	Pro-279 to Ala-286,	Pro-323 to Cys-328,	Asn-362 to Leu-367,
AA	SEQ	NO: Y	711	712	1070	713		1011	1072	1073	714	•		1074						1075		715								
	ORF	X (From-10)	159 - 527	127 - 267	117 - 257	123 - 323		116 - 307	1525 - 1566	345 - 665	158 - 430			153 - 536	17.07					212 - 484		184 - 2313								
	SEQ.		96	26	455	86		456	457	458	66			459						460		100								
	Contig		731863	1037893	895711	1043263		903816	905414	732097	1309174			1040056						882768		1352280								
	Gene CDNA Clone	3	HDPOJ08	HDPPN86	HDPPN86	HDPSB18		HDPSB18	HDPSB18		Г			HDPSH53			_			HDPSH53		HDPSP01		-						
	Gene		98	87		88					68											8								

OMIM Disease Reference(s):			104770, 107670, 110700, 145001, 146760, 146790, 191315, 601412, 601652, 601863, 602491															120260, 138140, 178300, 246450							
Cytologic Band			1921.2														•	1p33							
Predicted Epitopes	Thr-390 to Arg-397, Leu-490 to Arg-495, Gln-556 to Leu-561,	Gln-657 to Val-674. Gln-75 to Cvs-80.	Pro-29 to Lys-37.		Ci 12 to T 26	Gly-12 to 1 yr-20,	Val-52 to Asp-59,	Gln-88 to Asp-93,	Arg-124 to Asn-129,	His-193 to Arg-198,	Gln-207 to Thr-213,	Gln-338 to Arg-346,	Ser-378 to Ala-384,	Ser-413 to Arg-420,	Ser-428 to Glu-434,	His-443 to Ser-451,	Glu-454 to Ser-461.	Asp-57 to Glu-62,	Thr-91 to Ala-96,	Thr-114 to Ser-131,	Gly-133 to Pro-160,	Gln-356 to Arg-365,	Pro-383 to His-391,	Leu-401 to Trp-406,	Pro-430 to Asp-436.
SEQ ID NO. Y		9/01	1	1077	12.0	-						,						718							
SEQ ORF ID NO: (From-To)		227 - 1153	2356 - 2499	179 - 343	CLC - 7/1	40 - 1440												67 - 1434							
SEQ ID NO:		461	101	462	3	707												103							
Contig ID:		680120	744440	502472	217200	812/3/							- 7					1036997							
cDNA Clone ID		HDPSP01	HDPSP54	HDPSP54	PC IC IOII	HDPUW68												HDPVW11							
Gene No:			91		[92								_				93							

OMIM Disease Reference(s):		109270, 109270, 109270, 109270, 109270, 120150, 120150, 120150, 120150, 148065, 148080,	154275, 171190, 185800, 221820, 249000,	253250, 600119, 600119, 600525, 601844																								
Cytologic Band		17q21.33													-					٠								
Predicted Epitopes	Asp-57 to Gly-64.	Pro-36 to Ser-52, Ala-63 to Pro-78.	Ala-106 to Lys-115,	Glu-134 to Glu-141,	Val-155 to Asp-164,	Phe-199 to Gly-204,	Arg-218 to Leu-228,	Glu-230 to Val-235,	Val-247 to Pro-253,	Arg-262 to Gly-276,	Thr-303 to Gln-310,	Arg-335 to Trp-342,	Glu-399 to Ala-415,	Ser-458 to Glu-466,	Arg-508 to Asp-517,	Glu-580 to Pro-585,	Gln-620 to Trp-628,	Lys-651 to Ala-657,	Gly-677 to Met-682,	Ala-712 to Leu-717,	Gly-724 to Thr-731,	Arg-770 to Gln-775.	Pro-36 to Ser-52,	Ala-63 to Pro-78,	Ala-106 to Lys-115,	Glu-134 to Glu-141,	Val-155 to Asp-104.	
AA SEQ ID	1078	719							_														1079				900	1080
ORF : (From-To)	50 - 349	45 - 2453		-																			35 - 679				9	27 - 158
	463	104																					464					465
Contig ID:	896530	992925		-																			887914					905983
cDNA Clone ID	HDPVW11	HDPWN93																					HDPWN93					HDPWN93
Gene No:		94																										

	OMIM Disease Reference(s):														193300, 193300, 227646														_
	Cytologic Band	17													3p25.1						!						10p15.1		
	Predicted Epitopes	Pro-39 to Trp-44.	Pro-39 to Trp-44.			Arg-26 to Lys-46,	Ala-70 to Lys-81,	Gln-100 to Pro-105,	Val-118 to Leu-123,	Pro-166 to Pro-171,	Gly-310 to Gly-331.	Arg-26 to Lys-46,	Ala-70 to Lys-81,	Phe-92 to Gly-98.	Glu-91 to Arg-117,	Lys-124 to Ser-136,	Tyr-191 to Glu-200,	Glu-265 to Lys-272.	Glu-91 to Arg-117,	Lys-124 to Ser-136.	Glu-25 to Gly-31,	Tyr-62 to Thr-68,	Ala-189 to Glu-197,	Ala-204 to Gln-219.		Tyr-62 to Thr-68.	Lys-5 to Lys-10,	Asn-33 to Lys-39,	ASD-40 (0 LVS-24.
AA	SEQ 15 V	720	1081	1082	1083	721						1084	_		722				1085		723				1086		724		
	ORF O: (From-To)	23 - 319	33 - 329	539 - 607	1190 - 1267	274 - 1266						259 - 1257			288 - 1385				292 - 1389		70 - 729				65 - 727		326 - 2149		_
	SEQ ID NO: X	105	466	467	468	106						469			107				470		108				471		109		
	Contig ID:	879048	904768	895716	895715	1309175						834692			972757				906342		1307742				543618		785879		
	Gene cDNA Clone No: ID	HDPXY01	HDPXY01	HDPXY01	HDPXY01	нронр03						нронроз			HDTBD53				HDTBD53		HDTBP04				HDTBP04		HDTBV77		
	Gene No:	95				96									97						86						66		

OMIM Disease Reference(s):																							
Cytologic Band							X															18	
Predicted Epitopes	Pro-62 to Asp-67, Asn-116 to Arg-123, His-157 to Ala-162.	Val-242 to Lys-249, Val-251 to Asp-264.	Arg-24 to Arg-31,	ue-55 to 11p-41, Met-43 to His-52.	Arg-24 to Arg-31, Ile-33 to Gly-41.	Arg-24 to Arg-31.				Tyr-41 to Pro-46.	Leu-9 to Tyr-15,	Asp-34 to Gln-46,	Pro-51 to Asp-57,	Gly-88 to Thr-104,	I hr-123 to Ser-128.	Leu-31 to Asn-38.			Ala-84 to Gln-93.		Arg-28 to Gly-34.		Pro-43 to Cys-52,
AA SEQ ID NO: Y			725		1087	1088	726	1089	1090	727	728					1091	729	730	731	732	733	734	735
SEQ ORF ID NO: (From-To)			132 - 302		148 - 471	148 - 369	260 - 349	251 - 340	101 - 343	386 - 535	808 - 2427					515 - 757	57 - 209	116 - 241	99 - 398	28 - 228	237 - 341	91 - 309	201 - 953
SEQ ID NO:			110		472	473	111	474	475	112	113					476	114	115	116	117	118	119	120
Contig ID:			1306984		879009	751707	1043391	874477	892317	635457	619852					382025	545008	396139	740750	570903	411998	847060	834913
Gene cDNA Clone No: ID			ното раз		ното рез	HDTD023	HDTFE17	HDTFE17	HDTFE17	HDTGC73	HE2DE47					HE2DE47	HE2EN04	HE2FV03	HE2NV57	HE2PH36	HE6EU50	HE8DS15	HE8UB86
Gene No:			100				101			102	103						104	105	106	107	108	109	110

OMIM Disease Reference(s):					All the first the second secon																							
Cytologic Band			10																									
Predicted Epitopes	Lys-105 to Ser-113.	Pro-35 to Phe-41.	Asp-40 to Tyr-46.	Ser-39 to Asn-45,	Asn-103 to Ser-109.	Phe-31 to Asp-38,	Asn-59 to Tyr-65,	Ser-76 to Glu-82,	Thr-96 to Cys-108,	Gln-111 to Asn-118.	Gly-25 to Leu-30,	Pro-40 to Ser-49,	Pro-74 to Ser-91,	Asn-97 to Cys-104,	Pro-115 to Phe-123,	Ser-125 to Ser-132.	Gly-25 to Leu-30,	Pro-40 to Ser-49,	Pro-74 to Ser-91,	Asn-97 to Cys-104,	Pro-115 to Phe-123,	Ser-125 to Ser-132.	Ile-40 to Cys-49,	Arg-52 to Cys-57,	Ser-94 to Trp-99,	Gly-105 to Gly-111.	Ile-40 to Cys-49,	Arg-52 to Cys-57,
AA SEQ ID NO: Y		736	737	738		739				•	740	•					1092						741				1093	
ORF O: (From-To)		35 - 160	380 - 538	51 - 467		213 - 656					146 - 625						226 - 705						52 - 417				133 - 498	
SEQ ID NO:		121	122	123		124					125						477						126				478	
Contig ID:		420063	846309	701802		777843					1307611						1047700						885637				769649	
cDNA Clone ID		HE9HY07	HE9NN84	HEBEJ18		HEEAQ11					HEEB105						HEEBI05						HEGAN94				HEGAN94	
Gene No:		111	112	113		114					115											_	116					

OMIM Disease Reference(s):													141750, 141800, 141800, 141800, 141800,	141850, 141850, 141850, 141850, 141850,	156850, 186580, 191092, 600140, 600273,	601785											
								•					141750,	141850,	156850,	601313, 601785											
Cytologic Band		20p12.1											16p13.3														
Predicted Epitopes	Ser-94 to Trp-99, Gly-105 to Gly-111.		Met-1 to Pro-6,	Glu-58 to Cys-63,	Glu-65 to Gly-72,	Thr-74 to Asn-88,	Tyr-104 to Trp-109.	Met-1 to Pro-6,	Glu-58 to Cys-63,	Glu-65 to Gly-72,	Thr-74 to Val-87.		Pro-35 to Trp-42,	Ala-53 to Asp-62,	Arg-103 to Phe-110,	Ile-114 to Glu-120.	Pro-35 to Trp-42,	Pro-65 to Asp-72,	Thr-86 to Phe-93,	Ile-97 to Glu-103.	Pro-35 to Trp-42,	Pro-65 to Asp-72,	Thr-86 to Glu-92,	Pro-96 to Gly-104,	Ser-138 to Gly-154.		Ile-23 to Ala-29.
AA SEQ ID NO: Y		742	743					1094				744	745				1095				1096					746	747
SEQ ORF ID NO: (From-To)		123 - 266	73 - 438					67 - 435				198 - 332	25 - 411				62 - 397				57 - 524					60 - 197	161 - 355
SEQ ID NO: X		127	128					479				129	130				480				481					131	132
Contig ID:		603533	1307790					570048				598018	1352368				884824				748227					566811	703243
cDNA Clone ID		HEOMQ63	HEPAB80					HEPAB80				HEQAK71	HEQCC55				HEQCC55			-	HEQCCSS	•				HERAR44	HETBR16
Gene No:		117	118									119	120													121	122

SEQ	SEQ ORF	AA SEQ	Predicted Epitopes	Cytologic	MIMO
(r ro	m-10)	NO: Y		Dand	Disease Reference(s):
133 199	199 - 549	748			
134 232	232 - 492	749	Leu-69 to Leu-74.		
135 559	559 - 741	750	Gln-53 to Thr-60.		
136 47 - 1105	105	751	Ala-27 to Ser-38,	12q24	113100, 124200, 147440, 158590, 160781,
			Pro-43 to Asn-54,		163950, 163950, 251170, 276710, 600175,
			Thr-115 to Asp-121,		601517
			Leu-225 to Val-232,		
			Pro-247 to Gly-252, Arg-306 to Leu-311.		
487 - 519	613	752			
138 44 - 181	81	753	Lys-13 to Asn-19, Asn-27 to Asn-35.	4q32-q34	189800, 208400, 231675
139 1019 - 1135	135	754		Xp22.2	300075, 300077, 301200, 302350, 302801, 195435, 306000, 306000, 307800, 30800
					309510, 311200, 312040, 312170, 312700, 313400
140 24 - 167	7	755		22	
482 74 - 217	17	1097			
\dashv	84	756	Gln-31 to Pro-39.		
142 137 - 361	361	757	Lys-60 to Ser-74.		
	25 - 180	758		12,12p13	
	15 - 68	1098			
144 414 - 809	608	759	Pro-43 to Pro-50,		
_			Asn-65 to Gly-70.		
145 185 -	185 - 1834	760	Glu-25 to Lys-33,		
			Glu-115 to Lys-120,		
			Leu-162 to Cys-169, Glu-193 to Ile-203,		

Gene	Gene cDNA Clone	Contig	SEQ	ORF	AA SEQ	Predicted Epitopes	Cytologic	OMIM
Š	a		io X	ID NO: (From-To)	NO: Y		Band	Disease Reference(s):
						Ala-219 to Pro-225, Glu-261 to Thr-271		
						Lys-331 to Trp-336,		
						Lys-353 to Gly-358,		
				-		Phe-412 to Asp-417,		
						Gln-458 to Gly-467, Phe-533 to Gln-538		
	HFPCX09	835390	484	249 - 1895	1099	Glu-25 to Lys-33,		
	UCDUADO	508773	786	195 295	1100	Glu-113 to Lys-120.		
138	4-	520368	146	178 - 342	761	Pro-49 to Glv-54		
3 5	1	545012	147	158 - 262	762	Ala-19 to Lvs-34.	4012	103600, 103600, 103600, 104150, 104150.
<u>.</u>			:		2		ļ.	104500, 164920, 164920, 164920, 170650,
							•	006009
138	HFTDL56	926569	148	93 - 1652	292	Met-1 to Pro-7,		
						Gln-21 to Glu-27,		
						Arg-35 to Asp-49,		
						Asn-66 to Leu-72,		
						Trp-82 to Glu-95, Pro-158 to Asn-163.		
139	HFTDZ36	545726	149	547 - 753	764		16q24.3	155555, 155555, 227650, 253000, 602783
<u>동</u>	L	799525	150	114 - 284	765	Glu-44 to Asp-50.		
141	HFXAM76	601402	151	213 - 452	99/	Arg-30 to Gly-42,		
]	4	00000	ļ	0,7	1	Asp-30 to 3et-03.		
142	HFXBL33	778070	152	152 - 640	/0/			
143	HFXGT26	745381	153	13 - 270	292	His-56 to Gln-65, Leu-80 to Ile-85.		
14	HFXJU68	1352218	154	141 - 626	769		1p33	120260, 138140, 178300, 246450

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	Disease	(6)2000		120550, 120570, 120575, 121800, 130500, 133200, 133200	255800	100710	120260, 138140, 178300																			180105, 222800		
	Cytologic Band			1p34.1		17p11.1	1p32.2					11.01	20911.21													7933	7	
	Predicted Epitopes			Leu-16 to Ser-23,	Gly-53 to Leu-60.		Pro-10 to Arg-15,	Leu-96 to Ser-103,	Gly-172 to Pro-178,	Gln-213 to Asp-218,	Asn-268 to Leu-275,	AUS-202 to 1 116-209.	Ser-67 to Glu-74,	Arg-81 to Val-86,	Tyr-147 to Asp-160.	Ser-67 to Glu-74,	Arg-81 to Val-86,	Tyr-147 to Asp-160.	Ser-3 to Gln-10,	Val-14 to Gln-19,	Asp-32 to His-40,	Gly-50 to His-55,	Pro-76 to Ser-87.	Ser-40 to Gly-45,	Leu-73 to Arg-80.	Lys-74 to Tyr-79.		Asp-26 to Leu-36,
ΨV	SEQ ID	1011	770	771		772	773		-			, ;	774			1102			1103					775		176	777	778
	ORF (From-To)	486 148 - 348	98 - 241	204 - 443		273 - 422	87 - 965			-			14 - 1144			28 - 540			2 - 454					231 - 596		569 - 823	63 - 191	143 - 295
	SEQ ID NO:	486	155	156		157	158					3,	159			487			488					160		191	162	163
	Contig ID:	570855		069859		422794	570262					00000	837220			838602			899864					566838		494099	493724	562772
	Gene cDNA Clone No: ID	HFXJU68	HFXJX44	HFXKT05		HGBF079	HGBHI35						HGBIB74			HGBIB74			HGBIB74					HGLAF75		HHEMA75	HHENK42	HHENV10
	Gene No:		145	146		147	148						149											150		151	152	153

			_			_														_		_				<u> </u>			
MINO	OMILM Disease Reference(s):		120070, 120131, 120131, 138030, 259900																					180200, 180200, 180200, 180200, 600631					
	Cytologic Band		2q36.1					8	17									·				-		13q14.12				15,X	
	Fredicted Epitopes	Leu-42 to Phe-50.	Met-1 to Thr-13,	Ser-27 to Phe-34,	Arg-53 to Pro-59,	Ser-77 to Ser-82.	Pro-32 to Ser-39.				Ser-39 to Ser-44.	Tyr-39 to Arg-51.	Met-98 to Gln-107,	Gly-120 to Gly-126,	Pro-138 to Trp-145,	Leu-159 to Gly-169,	Val-211 to Arg-217,	Cys-256 to His-262,	Glu-320 to Val-327,	Phe-399 to Asn-406,	Asp-444 to Ser-450,	Asp-475 to Trp-488.		Ala-28 to His-41,	Pro-43 to Gln-64.	Trp-29 to Gly-42,	Gly-46 to His-51.	Thr-26 to Asn-39.	Pro-57 to Pro-64.
AA	NO: Y		611				780	781	782	1104	783	784	785										982	787		788		789	1105
40	O: (From-To)		269 - 517				192 - 530	230 - 361	270 - 536	270 - 302	107 - 241	71 - 238	183 - 1709										90 - 260	74 - 307		557 - 712		291 - 425	50 - 439
	N NO		164				165	166	167	489	168	169	170										171	172		173		174	490
	Contig ID:		877639				411332	340818	662329	383547	554613	695726	695134										553494	456466		487807		895505	821341
	Gene cDNA Clone No: ID		HHEPM33				HHFHJ59	HHGCG53	HHGCM76	HHGCM76	HHGDW43	HIPEC09	HHPEN62										HHSDX28	HJABB94		HJABX32		HJACG30	HJACG30
	Gene No:		154				155	156	157		158	159	160										191	162		163		164	

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OMIM Disease Reference(s):								144120 147020 147110			120950 120960 130500 133200 138140	168360 171760 171760 176100	178300, 187040, 230000, 255800, 600101.	600650, 600650, 600722, 600722													
Cytologic Band		7p22.3						14032 33			1n32-n34	- Land															
Predicted Epitopes	Lys-1 to Gly-8.	Glu-35 to His-41,	Ser-62 to Ala-67,	Pro-145 to Leu-155,	Glu-157 to Ser-163,	Arg-190 to Val-197,	Asp-208 to Pro-215,	127 0110 127 120	Dr. 42 to Cu. 50	1 eu-61 to Ala-66.	Phe-25 to Ser-30				Ser-25 to Ala-31,	Gln-146 to Ser-151,	His-231 to Asn-236.	Ser-25 to Ala-31,	Gln-146 to Ser-151,	His-231 to Asn-236.	Tyr-39 to Lys-58.	Thr-42 to Pro-53,	Val-78 to Glu-86,	Glu-103 to Met-112,	Ala-124 to Gly-131,	Trp-158 to Glu-168,	Gln-189 to Phe-210,
AA SEQ ID NO: Y	+-	790						701	T		703				794			1107			262	962					
ORF O: (From-To)	350 - 715	232 - 1215						348 - 518	60 225	ccc - 00	714 417	111-117			77 - 808			008 - 69			<i>27 - 2</i> 69	38 - 940					
SEQ ID NO:	491	175						176	222	\	178	2			179			492			180	181					
Contig ID:	774300	719729						568878	70000	/65150	86038	2/200			862030			665424			554616	1352202					
Gene cDNA Clone No: ID	HJACG30	HJBCY35						HIMBNSO	SECTABLE 1	FUFAD/3	LIV A B19A	Fordown			HKABZ65			HKABZ65			HKACB56	HKACD58					
Gene No:		165						1,66	3 5) OI	1,60	20			169						170	171					

OMIM Disease Reference(s):					151570 601790	10100, 001100		
Cytologic Band					15,000	7:77601		
Predicted Epitopes	Ala-221 to Gly-226, Arg-274 to Asp-284, Ala-294 to Gly-299.	Thr-42 to Pro-53, Val-78 to Glu-86, Glu-103 to Met-112,	Ala-124 to City-131. Cys-31 to Arg-36, Asp-81 to His-86,	Thr-6 to Trp-13, Thr-75 to Gln-80, Thr-112 to Tyr-117, Leu-133 to Pro-138, Ala-146 to Phe-153, Gln-319 to Ser-325, Val-354 to His-372, Pro-391 to Gly-396, Val-405 to Thr-412, Ile-425 to Asp-437.	Thr-6 to Trp-13.	Ser-51 to Thr-57.	Ser-51 to Thr-57. Gln-23 to Asp-28.	Ser-7 to Pro-14, Arg-47 to Arg-52, His-117 to Val-123,
AA SEQ ID NO: Y		1108	797	798	1109	800	1110	801
ORF ORF (From-To)		35 - 499	229 - 1056	501 - 1814	197 - 370	508 - 831	508 - 831 234 - 347	178 - 879
SEQ ID NO: X		493	182	183	494	185	495 496	186
Contig ID:		552465	604123	1352263	638238	946512	889258 904790	876571
Gene CDNA Clone		HKACD58	НКАDQ91	HKAEV06	HKAEV06	HKAFT66	HKAFT66 HKAFT66	HKB1E57
Gene No:			172	173		175		176

OMIM Disease Reference(s):							300047, 300071, 300110, 300600, 301000, 301000, 301830, 309470, 309500, 309610, 309850, 311050, 312060
Cytologic Band							Xp11.23
Predicted Epitopes	Glu-142 to Thr-149, Leu-162 to Ala-167, Gly-172 to Asn-177, Thr-226 to Ala-232.	Met-1 to Tyr-6, Thr-38 to Ala-44.	Arg-52 to Ala-58, Thr-121 to Lys-126, Gly-156 to Gln-164, Gly-201 to Glu-215, Thr-432 to Gly-450, Glu-461 to Gly-466.	Ala-28 to Ala-33, Arg-38 to Leu-48, Thr-120 to Lys-125, Gly-155 to Gln-163, Gly-200 to Glu-214.	Ala-1 to Gly-6, Ala-10 to Tyr-18. Ala-1 to Gly-6.	Ala-10 to Tyr-18. Lys-23 to Lys-29.	Pro-36 to Gly-42, Gly-54 to Arg-65, Ala-85 to Ala-91, Ala-95 to Gln-102, Ala-115 to Pro-121,
AA SEQ ID NO: Y		1112	802	1113	1114		
SEQ ORF ID NO: (From-To)		30 - 170	64 - 1473	41 - 1369	3 - 929	313 - 591	53 - 835
SEQ ID NO: X	_	497	187	498	499	188	189
Contig ID:		654871	1352286	701893	513190	601969	877489
Gene CDNA Clone No: ID		HKB1E57	HKFBC53	HKFBC53	HKFBC53 HKFBC53	HKGC027	HKGDL36
Gene No:			177			178	179

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OMIM Disease Reference(s):						101000, 101000, 101000, 101000, 123620,	138981, 188826, 600850, 601669				118800, 123660, 125660, 125660, 193500,	193500, 193500, 193500, 201460, 205100, 237300, 262000, 600266, 601277		•			118485, 151670, 231680, 272800, 272800, 272800, 276700, 600374, 601780			5p15.2-p14.1 123000, 602568		126090, 129010, 142600, 154545, 250850,
Cytologic Band						22q12.2					2q35						15q23			5p15.2-p14.	•	10q21-q22
Predicted Epitopes	Pro-166 to Asp-191, Lys-243 to Ala-249.	Pro-36 to Gly-42,	Pro-64 to Ala-76,	Gly-83 to Ala-90,	Ser-100 to Cys-108, Thr-126 to Ser-135.	Ala-23 to Arg-36,	His-38 to Ala-46,	Pro-50 to Gly-56,	Arg-85 to Val-94.	Ala-66 to Leu-/3.	Gly-27 to Cys-35.		Ala-59 to Thr-68	Glu-72 to Ser-108,	Glu-115 to Lys-126.		Arg-28 to Gln-36.	Pro-171 to Gln-179,	Leu-218 to Lys-225, Phe-266 to Cys-275	Arg-122 to Ser-139,	Met-144 to Glu-149.	Leu-68 to Lys-74,
AA SEQ ID NO: Y		1117				805			, is	800	807		808	3		608	810	811		812		813
SEQ ORF ID NO: (From-To)		55 - 501				130 - 417			0.00	43 - 2/3	20 - 229		87 - 474	1		202 - 327	368 - 709	43 - 870		520 - 1005		99 - 1142
SEQ ID NO: X		502				190			ļ	191	192		103			194	195	196		197		198
Contig ID:		704088				625956			4,000	580845	587269		\$14788	200		281399	636083	847396		753742		740755
cDNA Clone ID		HKGDL36				HKISB57				HKIYP40	HKMLK53		HKMI M11			HKMMW74	HLDON23	HLDOW79		HLDQR62	,	HLDQU79
Gene No:						180			į	181	182		183	3		184	185	186		187		188

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OMIM Disease Reference(s):								3p21.2-p21.3 116806, 120120, 120120, 120436, 120436, 138320, 168468, 182280.	238310, 600163, 601226											
Cytologic Band								3p21.2-p21.3					17							
Predicted Epitopes	Gin-200 to Val-205, Lys-207 to Lys-214, Glu-237 to Ile-244,	Ala-271 to Thr-279, Ser-317 to Ser-329, Gln-342 to Gly-348.	Leu-32 to His-38.	Tyr-28 to Phe-34,	Thr-54 to Val-60, Tvr-73 to Thr-82.	Met-37 to Ser-43.	Pro-55 to Gly-66, Phe-92 to Leu-103.						Gly-4 to Glu-9,	Asp-22 to Cys-28,	Glu-39 to Leu-44,	Gly-4 to Glu-9.	Gly-1 to Glu-8,	Gly-37 to Gly-61,	Gln-71 to Phe-81,	Asp-95 to Gly-103,
AA SEQ ID NO: Y			814	815		816	817	818		819	820	821	822			1118	1119			
SEQ ORF ID NO: (From-To)			30 - 164	224 - 574		186 - 338	249 - 869	205 - 381		74 - 160	155 - 280	5 - 232	226 - 516			226 - 423	3 - 899			
SEQ ID NO: X			199	200		201	202	203		204	202	907				503	504			
Contig ID:			684216	460467		778073	791828	588446		520231	396672	699812	1087335			1035443	1047690			
cDNA Clone ID			HLHAL68	HL.HFP03		HLIBD68	игісбэо	нгорн79		HLTDV50	HLTE125	HLTHR66	HLTIP94			HLTIP94	HLTIP94			
Gene No:			189	190		161	192	193		194	195	196	197							

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МІМО	Disease Reference(s):		104770, 107670, 110700, 135940, 145001,	146790, 152445, 152445, 159001, 174000,	179755, 182860, 182860, 182860, 191315,	230800, 230800, 266200, 600897, 601105,	601412, 601652, 602491		107777, 123940, 139350, 139350, 148040,	148041, 148043, 148070, 231550, 600194,	600231, 600536, 600808, 600956, 601284,	601769, 601769, 601928, 602116, 602153		125370, 601458											222900, 601402			
Cytologic	Band		1921	_					12q13					12p13.31											3q25.1			
Predicted Epitopes		Leu-126 to Ile-131, Val-166 to Glu-171.	Lys-17 to Glu-27,	Gln-40 to Gly-47.					Cys-126 to Thr-138,	Glu-165 to Gly-172,	Thr-189 to Leu-200,	Gly-222 to Gly-229,	Pro-346 to Lys-354.	Asp-27 to Ser-32,	Pro-52 to Thr-58,	Arg-63 to Asn-70,	Gln-78 to Gly-83,	Thr-107 to Asn-113,	Thr-160 to Val-176,	Ser-188 to Gly-241,	Leu-248 to Pro-265,	Tyr-302 to Gly-314.			Asp-59 to Asn-65,	Lys-72 to Trp-79,	Tyr-110 to Val-121,	Ala-204 to Leu-216.
AA SEO	NO: Y		823					824	825					826									827	828	829			
ORF	<u> </u>		436 - 996					326 - 748	212 - 1276					38 - 1054									155 - 328	92 - 232	190 - 855		•	
SEO	ID NO:		208					209	210					211									212	213	214			
Contie	iii		629552					653513	587270					658702									460619	778075	1352163			
cDNA Clone	Ð		HLWAA17					HLWAD77	HLWA022	_				HLWAY54									HLWCF05	HLYAC95	HLYAZ61			
Gene	No.		198					199	200					201									202	203	204			

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	OMIM Disease Reference(s):		138760, 186580, 249100, 266600, 600760, 600760, 600761, 600761	182600, 186880, 190195, 190195, 222700, 600243, 602279, 602279																				
	Cytologic Band		16p13	14q11.2																		_		
	Predicted Epitopes	Asp-59 to Asn-65, Lys-72 to Trp-79, Tyr-110 to Val-121, Ala-204 to Asn-215.	Gly 43 to Gly-55.	Arg-48 to Asn-56, Gly-166 to Ser-175,	Tyr-250 to Leu-261,	Glu-529 to Gly-555, Ala-378 to Tvr-383,	Gly-390 to Tyr-413,	Pro-422 to Cys-433,	Gln-491 to Tyr-496,	Phe-511 to Ser-520,	Pro-542 to Arg-551,	Arg-568 to Val-582,	Gly-595 to Glu-601,	Gln-608 to Pro-614,	Pro-669 to Pro-678.	Arg-48 to Asn-56.	Gly-33 to Lys-41,	Pro-52 to Lys-60,	Asn-81 to Ala-86,	Lys-156 to Met-164,	Gln-283 to Lys-292,	Glu-303 to Gly-308.	Gly-33 to Lys-41,	Fro-52 to Lys-60,
AA	SEQ ID NO: Y	1120	830	831												1121	832						1122	
	SEQ ORF ID NO: (From-To)	205 - 852	161 - 619	491 - 2629												115 - 348	4 - 1023						3 - 923	
	SEQ ID NO: X	505	215	216		•										506	217						202	
	Contig ID:	423998	561941	1352177												467053	1352406						1049263	
	cDNA Clone ID	HLYAZ61	HMADK33	HMADU73												HMADU73	HMAMI15						HIMAMI15 .	
	Gene No:		205	206													207							

OMIM Disease Reference(s):												602092				278700, 602088								-				
Cytologic Band												11p14.3				9q22.33				٠								
Predicted Epitopes	Asn-81 to Ala-86.			Pro-53 to Thr-65.		Gln-85 to Lys-91,	Pro-106 to Ser-117,	Pro-124 to Ala-130,	tip-15- to tip too:	000	Ser-34 to Ser-39.	Pro-18 to Lys-26.		Ser-20 to Ser-34,	Thr-40 to Ser-46.	Glu-78 to Asn-83,	Asp-91 to Gln-100,	Glu-122 to Ser-128,	Arg-137 to Pro-143,	Asp-157 to Asn-162,	Glu-168 to Asn-174,	Ser-199 to Gly-206,	Pro-213 to Ala-218,	Glu-251 to Thr-257,	Ser-353 to His-361,	Gly-363 to Ala-375,	Pro-382 to Phe-387,	Arg-401 to Leu-406.
AA SEQ ID NO: Y		833	834	835	836	837	•		070	000	839	840	841	842		843												
ORF (From-To)		175 - 369	273 - 407	180 - 428	50 - 211	34 - 699			227 451	332 - 431	92 - 280	49 - 342	221 - 370	142 - 294		157 - 1377								-				
SEQ ID NO: X		218	219	220	221	222			200	577	224	225	526	227		228												
Contig ID:		635301	929095		636035	260775			20000	320307	520304	603201	548078	562776		1309723												
Gene cDNA Clone No: ID		HMCFY13	HMDAB56	HMDAQ29	HMECK83	HIMEED18			TOWNER	FINEF 134	HIMEGF92	HMIAL37	HMKCG09	HMMAH60		нморт36												
Gene No:		208	509	210	211	212			51,0	713	214	215	216	217		218												

OMIM Disease Reference(s):							
Cytologic Band		d£'£					
Predicted Epitopes	Glu-78 to Asn-83, Asp-91 to Glu-100, Glu-122 to Ser-128, Arg-137 to Pro-143, Asp-157 to Asn-162, Glu-168 to Asn-174, Ser-199 to Gly-206, Pro-213 to Ala-218, Glu-251 to Thr-257, Ser-353 to His-361, Gly-363 to Ala-375, Pro-382 to Phe-387, Arg-401 to Leu-406.	Ser-31 to Lys-45, Pro-47 to Pro-53, Ser-58 to Arg-63.	Ser-31 to Lys-45, Pro-47 to Pro-53, Ser-58 to Arg-63.			Thr-28 to Arg-49, Ser-57 to Arg-64, Pro-72 to His-78.	Thr-28 to Arg-49, Ser-57 to Arg-64.
AA SEQ ID NO: Y	1123	844	1124	1125	845 846	847	1127
SEQ ORF ID NO: (From-To) X	192 - 1412	531 - 725	528 - 722	565 - 645	120 - 308 28 - 141	134 - 445	162 - 473
SEQ ID NO: X	809	229	509	510 511	230	232	512
Contig ID:	424085	973996	895429	904241 750927	560229 545427	1127691	1028961
Gene cDNA Clone No: ID	HMQDT36	HMSDL37	HMSDL37	HMSDL37 HMSDL37	HMSF126 HMSFS21	HMSHS36	HMSHS36
Gene No:		219			220	222	

OMIM Disease Reference(s):			102200, 106100, 131100, 131100, 131100, 133780, 147050, 153700, 161015, 164009, 168461, 168461, 168461, 180721, 180840, 191181, 193235, 209901, 232600, 259700, 259770, 600045, 600319, 600528, 601884						
Cytologic Band			11913						
Predicted Epitopes	Thr-27 to Arg-33, Gly-37 to Ser-42, Pro-52 to Arg-72.	Pro-43 to Leu-49, Pro-61 to Gly-66, Ser-71 to Ser-83.						Pro-18 to Gly-30, Arg-98 to Cys-103, Glu-106 to Arg-111, Ser-117 to Gly-122, Glu-132 to Ala-140, Pro-247 to Arg-252, Val-301 to Ala-308, Pro-334 to Ser-339, Arg-348 to Thr-354, Glu-427 to Gly-439, Gly-442 to Glu-448,	Pro-18 to Gly-30.
AA SEQ ID NO: Y	848	849	850	851	852	853	1128	854	1129
ORF (From-To)	133 - 354	306 - 560	34 - 453	124 - 252	72 - 437	367 - 456	129 - 185	42 - 1514	42 - 608
SEQ ID NO: X	233	234	235	236	237	238	513	239	514
Contig ID:	799540	588447	639203	460487	562063	825421	490495	1308287	794987
cDNA Clone ID	HMSKC04	HMTAD67	HMVBS81	HMWDC28	HMWFT65	HIMWFY10	HIMWFY10	HMWGY65	HMWGY65
Gene No:	223	224	225	226	227	228		229	

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OMIM Disense Reference(s):		120140, 120140, 120140, 120140, 120140, 120140, 120140, 126337, 600808, 601284, 601769, 601769, 602116																					
Cytologic Band		12q13.12								10,C					-		11						
Predicted Epitopes		Asp-21 to Ser-29.		Pro-47 to Met-53, Ser-130 to Ser-138.	Val-25 to Gly-33.			Met-1 to Arg-8, Leu-35 to Glu-41.			Pro-18 to Glu-25.	Ala-28 to Gly-34, Pro-57 to Thr-66.				Asn-46 to Ser-54.	Met-1 to Gly-9.	Met-1 to Gly-9.					
AA SEQ ID NO: Y	855	856	857	858	829	860	861	862	863	864	865	998	867	898	698	870	871	1130	1131	872	873	1132	1133
ORF O: (From-To)	213 - 428	488 - 691	170 - 409	228 - 929	86 - 286	275 - 478	50 - 151	98 - 232	108 - 380	178 - 300	135 - 245	221 - 433	77 - 217	321 - 545	436 - 597	388 - 636	27 - 200	27 - 200	296 - 877	328 - 492	52 - 162	28 - 138	166 - 252
SEQ ID NO: X	240	241	242	243	244	245	246	247	248	249	250	251	252	253	254	255	256	515	516	257	258	517	518
Contig ID:	553558	753337	460611	634551	577013	410107	520227	532622	553552	410179	519120	526651	561568	604891	834857	839224	1041375	838184	839283	496115	088586	902442	842223
cDNA Clone ID	HNEEE24	HNFFC43	HNFIU96	HNFIY77	HNFJF07	HNFJH45	HNGAP93	HNGE029	HNGFR31	HNGIH43	HNGIJ31	HNGIQ46	HNGJES0	HNGJP69	68NYDNH	HNGND37	HNGO112	71105NH	HNGOII2	HNHAH01	HNHE142	HNHE142	HNHEI42
Gene No:	230	231	232	233	234	235	236	237	238	239	240	241	242	243	244	245	246			247	248		

OMIM Disease Reference(s)	./6\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\																						116806, 168468, 182280, 212138, 600163		-	•	
Cytologic Band			1																				3p21.31				
Predicted Epitopes	Pro-10 to Cys-19.		Glu-67 to Ala-74.	Glu-17 to Lys-30,	Val-43 to Asn-53.			Pro-56 to Pro-63,	Met-92 to Thr-98,	Ser-112 to Pro-120,	Pro-162 to Glu-173,	Ala-200 to Ser-210,	Lys-311 to Asn-320.	Pro-56 to Pro-63,	Met-92 to Thr-98,	Ser-112 to Pro-120,	Pro-162 to Ser-169.	Pro-56 to Pro-63,	Met-92 to Thr-98,	Arg-107 to Pro-120.	Met-1 to Trp-15,	Thr-52 to Met-58.	Arg-45 to Thr-52,	Tyr-60 to Gly-66,	Ala-87 to Trp-92,	Leu-105 to Ser-115.	Tyr-2 to Gly-15, Tro-192 to Asp-199.
AA SEQ ID	1134	874	875	928		877	878	879						1135				1136			880	-	881				882
SEQ ORF ID NO: (From-To)	331 - 435	57 - 302	38 - 280	40 - 201		12 - 251	291 - 413	28 - 990						32 - 547				16 - 411			210 - 386		100 - 447				111 - 1316
SEQ ID NO:	519	259	260	261		262	263	264						520				521			265		266				267
Contig ID:	823723	634851	664507	895462		843488	834927	1310821						796807				590738			270877		545534				1160395
cDNA Clone ID	HNHEI42	HINHEU93	HNHFM14	HINHINB 29		HNHOD46	HNHPD10	HNTBI26						HNTB126				HNTB126			HNTBI57		HNTBL27				HNTCE26
Gene No:		249	250	251		252	253	254													255		256				257

OMIM Disease Reference(s):																									
Cytologic Band																									
Predicted Epitopes	Lys-248 to Leu-253, Arg-330 to Lys-336, Gln-354 to Val-364,	Val-383 to Ser-392. Arg-75 to Lys-81.	Gln-99 to Asp-109.	Lys-71 to Trp-76.		Thr-28 to Ser-40.			Lys-50 to Phe-57,	Ser-70 to Arg-77,	Tyr-81 to Ser-87,	Pro-112 to Thr-117.	Lys-39 to Phe-46,	Ser-59 to Arg-66,	Tyr-70 to Ser-76,	Pro-101 to Thr-106.	Thr-49 to Arg-54,	Leu-147 to Asp-153.	Leu-37 to Gly-44,	Thr-137 to Leu-144,	Ala-178 to Asn-184,	Asp-194 to Val-201,	Leu-252 to Glu-258,	Asp-280 to Tyr-293,	Asn-296 to Thr-301,
AA SEQ ID NO: Y		1137		883	1138	884	885	988	887				1139				888		688				-		
SEQ ORF ID NO: (From-To)		57 - 422		307 - 534	306 - 455	46 - 171	251 - 313	434 - 541	1 - 477				27 - 473	-			64 - 606		49 - 1503						
SEQ ID NO: X		522		268	523	269	270	271	272				524				273		274						
Contig ID:		853373		1352285	699848	684307	520348	422913	1194866				834999				833079		1184465						
cDNA Clone ID		HNTCE26		HNTNI01	HNTNI01	HODDF13	S9NGGOH	HODDN92	HODFN71				HODFN71				HOEFV61		НОЕМОЗЗ						
Gene No:				258		259	260	261	797								263		264						

OMIM Disease Reference(s):					-
Cytologic Band					
Predicted Epitopes	Asp-322 to Asp-348, Asn-363 to Ser-368, His-370 to Thr-378, Asn-380 to Cys-386, Glu-391 to Cys-399, Leu-421 to Arg-426, Glu-454 to Tyr-459.	Leu-37 to Gly-44, Pro-46 to Gly-51, Thr-137 to Leu-144, Ala-178 to Asn-184, Asp-194 to Val-201, Leu-252 to Glu-258, Asp-280 to Tyr-293, Asp-322 to Asp-348, Asp-322 to Asp-348, Asn-363 to Ser-368, His-370 to Thr-378, Asn-380 to Cys-386, Glu-391 to Cys-399, Leu-421 to Arg-426, Glu-454 to Tyr-459.	Leu-37 to Gly-43.	Met-2 to Ser-9.	Thr-30 to Met-36, His-121 to Thr-136, Leu-231 to Gly-236, Thr-248 to Pro-256,
AA SEQ ID NO: Y		1140	1141	1143	068
SEQ ORF (From-To)		48 - 1502	78 - 875 724 - 741	123 - 374	83 - 1315
SEQ ID NO: X		525	526 527	528	275
Contig ID:	·	919896	906694	702186	911180
Gene cDNA Clone		ноғм (33	HOFMQ33 HOFMQ33	НО БМО 33	HOFMT75
Gene No:					265

	OMIM Disease Reference(s):																											
	Cytologic Band																											
	Predicted Epitopes	Gly-342 to Thr-353.	Thr-30 to Met-36.		Thr-30 to Met-36,	Pro-51 to Ser-56,	His-121 to Thr-136,	Leu-233 to Gly-243,	Thr-250 to Ser-258,	Thr-265 to Trp-270.			Pro-22 to Cys-30,	Gly-43 to Tyr-53,	Ser-55 to Trp-65,	Ala-76 to His-81,	Pro-101 to Gly-108,	Pro-121 to Gly-127.	Thr-47 to Pro-55.	Pro-1 to Val-7.		Thr-60 to Ala-65,	Leu-94 to Glu-99,	Cys-182 to Trp-188.		Pro-33 to Phe-43,	Pro-48 to Lys-54, His-61 to Val-66	Arg-40 to Gly-45,
AA	SEQ ID NO: Y		1144	1145	1146						891	1147	892				•		1148	1149	1150	893			1151	894		895
	SEQ ORF ID NO: (From-To) X		83 - 427	1225 - 1500	129 - 1232						79 - 297	155 - 373	18 - 407						23 - 226	127 - 171	142 - 162	514 - 1254			1455 - 1472	232 - 831		136 - 771
	SEQ ID NO: X		529	530	531						9/2	532	277						533	534	232	8/2			536	579		280
	Contig ID:		905365	892308	892291						1352378	899292	931871						907073	907072	878863	088568			902295	625973		1299928
	cDNA Clone ID		HOFMT75	HOFMT75	HOFMT75						HOFNC14	HOFNC14	HOFOC73			a			HOFOC73	HOFOC73	HOFOC73	100СК63			HOGCK63	HOHBY12		HONAH29
	Gene No:										266		267									268				569		270

OMIM Disease Reference(s):													157147, 248510
Cytologic Band										-			4924
Predicted Epitopes	Leu-56 to Ser-61, Ser-65 to Gly-72, Gly-87 to Leu-92, Tyr-111 to Pro-121, Arg-135 to Phe-144,	Phe-188 to Asp-196.	Ser-30 to Met-36, Ile-38 to Pro-46,	Gln-78 to Gly-88, Thr-98 to Pro-105,	Gly-110 to Ser-122, Ser-136 to Trp-144.	Ser-30 to Met-36,	lie-38 to Pro-45, Gln-78 to Gly-88,	Thr-98 to Pro-105, Gly-110 to Ser-122.			Gly-18 to Lys-23, Pro-31 to Gly-38.	1155 Gly-18 to Lys-23, Pro-31 to Gly-38.	Asn-15 to Trp-20, Ser-36 to Gly-41, Pro-103 to Val-110,
AA SEQ ID NO: Y		1152	968			1153			1154	897	868	1155	899
SEQ ORF ID NO: (From-To)		144 - 779	361 - 852			102 - 584			55 - 1029	89 - 259	1076 - 1195	146 - 268	56 - 1927
SEQ ID NO: X		537	281			538			539	282	283	540	284
Contig ID:		457167	1352356			858338			857453	589431	854234	566845	614040
Gene cDNA Clone		HONAH29	ноовл82			НООВ182			HOQBJ82	HOSBY40	HOSD125	HOSDJ25	HOSFD58
Gene No:			271							272	273		274

OMIM Disease Reference(s):				120215, 120215, 190198	162150		
Cytologic Band				9q34.3	5q15		
Predicted Epitopes	Pro-134 to Arg-143, Leu-173 to Arg-178, Ser-190 to Ala-197, His-314 to Arg-319, Arg-354 to Asn-362, Asp-391 to Arg-397, Glu-402 to Asp-409, Asp-434 to Leu-439, Glu-441 to Arg-446, Gly-455 to Asp-462, Pro-528 to His-541, Asn-566 to Arg-571, Tyr-574 to Glu-581, Thr-589 to Glu-603.	Gly-28 to Leu-42, Met-52 to Leu-58.	Gly-8 to Leu-14, Met-18 to Phe-30.	Leu-20 to Ala-26, Arg-32 to Arg-39, Thr-104 to Gly-112.	Arg-29 to Pro-37, Gln-46 to Val-56.	Arg-29 to Pro-37, Gln-46 to Val-56.	Gln-51 to Thr-61, Ser-65 to Thr-71, Pro-85 to Gln-91.
AA SEQ ID NO: Y		1156	006	901	905	1157	903
ORF O: (From-To)		477 - 659	508 - 3408	86 - 445	51 - 446	510 - 905	203 - 496
SEQ ID NO: X		541	285	286	287	542	288
Contig ID:		383513	429229	411080	1306899	422936	520367
Gene cDNA Clone No: ID		85ДЫЅН	HOUCQ17	HPBCU51	HPDDC77	HPDDC77	HPEAD48
Gene No:			275	276	277		278

OMIM Disease																											
Cytologic Band																4,8				1p36.33							
Predicted Epitopes	Lys-16 to Ser-21, Gly-36 to Asp-41.		Asp-40 to Glu-50,	Ser-59 to Gly-69,	Leu-109 to Lys-117,	Tyr-130 to Leu-137,	Leu-140 to Glu-160,	Gly-202 to Tyr-208.	Asp-40 to Glu-50,	Ser-59 to Gly-69,	Ala-98 to His-105,	Arg-108 to Glu-114,	Pro-124 to Ser-138,	Ala-143 to Gly-154.	Arg-30 to Gln-36.					Ala-55 to Asn-60,	Lys-65 to Met-71,	Leu-75 to Asn-86,	Asp-93 to Asp-110,	Leu-130 to Cys-138,	Gln-149 to Glu-154,	Thr-172 to Ile-179,	Glu-185 to Arg-192.
AA SEQ ID		905	906						1158						206	806	1159	1160	1161	606							
SEQ ORF ID NO: (From-To)	51 - 176	79 - 126	128 - 763						127 - 648						236 - 397	126 - 272	119 - 265	1001 - 696	509 - 523	69 - 69							
'SEQ ID NO:	289	290	291						543						292	293	544	545	246	294							
Contig ID:	520202	519003	1310868						590741						682699	1011467	525375	796925	699587	846357							
cDNA Clone ID	HPEAD79	HPEBE79	HPIBO15						HPIB015						HPJB133	HPJBK12	HPJBK12	HPJBK12	HPJBK12	HPMDK28							
Gene No:	279	280	281												282	283				284							

					AA	;	•	
Gene	Gene cDNA Clone	Contig	SEQ	ORF	SEO	Predicted Epitopes	Cytologic	OMIM
So:	a		ID NO): (From-To)	<u>e</u>		Band	Disease
			X		NO: Y			Reference(s):
	HPMDK28	639118	547	58 - 663	1162	Ala-55 to Asn-60,		
						Lys-65 to Met-71,		
						Leu-75 to Asn-86,		
						Asp-93 to Asp-110.		
						Leu-130 to Cys-138,		
						Gln-149 to Glu-154,		
						Thr-172 to Ile-179.		
						Glu-185 to Arg-192.		
285	HPRAL78	1352342	295	62 - 1321	910	Pro-31 to Thr-48,	3p25.2	193300, 193300, 227646
						Arg-62 to Gly-70,		
						Ala-74 to Glu-87,		
						Lys-123 to Asp-129,		
						Pro-162 to Gly-167,		
						Glu-170 to Gly-189,		
	-					Arg-220 to Asn-228,		
						Glu-248 to Ala-258,		
						Gly-285 to Gly-300,		
						Pro-315 to Gly-327,		
_						Ser-406 to Arg-411.		
	HPRAL78	844216	548	70 - 1245	1163	Pro-31 to Thr-48,		
						Arg-62 to Gly-70,	-	
						Aia-/4 to Giu-8/,		
						Lys-123 to Asp-129,		
						Pro-162 to Gly-167,		
				•		Giu-1/0 to Giy-16%,		
						Arg-220 to Asn-228.		
	HPRAL78	484735	549	148 - 339	1164	Ser-49 to Arg-54.		
286	HPRBC80	829136	296	94 - 1254	911	Asp-6 to His-13,	2p21	120435, 120435, 126600, 135300, 136435,
						Asp-114 to Gly-131,		152790, 152790, 157170, 182601, 601771

OMIM Disease Reference(s):			103581, 146150, 176270, 218000, 227220, 601623, 601800, 601889, 602117	143890, 151440, 600276, 601843																					
Cytologic Band			15q11-q13	19p13.11		4																			
Predicted Epitopes	Thr-166 to Gln-181, Val-210 to Thr-216, Pro-222 to Tyr-227.			Ser-3 to Lys-8.						Pro-21 to Pro-26,	Arg-31 to Asn-37.	Pro-21 to Pro-26,	Arg-31 to Asn-37.	Pro-21 to Pro-26,	Arg-31 to Lys-37.	Lys-32 to Lys-38.	Asn-49 to Gln-54,	Glu-150 to Asp-159.	Ala-30 to Gly-36,	Asp-45 to Trp-50,	Lys-65 to Cys-71,	Pro-80 to Cys-87.	Ala-30 to Gly-36,	Asp-45 to Trp-50,	Lys-65 to Cys-/1,
AA SEQ ID NO: Y		1165	912	913	914	915	1166	1167	916	917		1168		6911		816	616		076				0/11		
SEQ ORF (From-To)		404 - 613	127 - 306	318 - 560	80 - 214	468 - 626	474 - 632	178 - 435	88 - 321	149 - 310		149 - 313		161 - 301		34 - 177	35 - 514		144 - 452		•		130 - 438		
SEQ ID NO: X		550	297	298	299	300	551	552	301	302		553		554		303	304		305				555		
Contig ID:		720095	526310	634353	526749	1001560	876469	789574	413270	722246		709662		692213		585702	658717		882176				588460		
cDNA Clone ID		HPRBC80	HPRSB76	HPTVX32	HPVAB94	HPWAY46	HPWAY46	HPWAY46	HPWAZ95	HPWDJ42		HPWDJ42		HPWDJ42		HPZAB47	HRAAB15		HRABA80				HRABA80		
Gene No:			287	288	289	290			291	292						293	294		295						

OMIM Disease Reference(s):				148900, 216550																								
Cytologic Band				8q22.2	ı														I									
Predicted Epitopes	Pro-80 to Cys-87.			Arg-31 to Lys-37,	Lys-58 to Glu-65,	Asp-157 to Gly-168,	Ile-219 to Gly-225,	Ala-260 to Ser-268,	A == 21 to 1 == 27	Arg-31 to Lys-3/,	Lys-58 to Glu-65,	Asp-157 to Gly-168,	Ile-219 to Gly-225,	Ala-260 to Ser-268,	Thr-276 to Glu-282.	Ile-9 to Gly-15,	Ala-50 to Ser-58,	Thr-66 to Glu-72.	Thr-48 to Arg-56,	Pro-122 to Glu-127,	Lys-135 to Cys-143,	Ala-180 to Gly-185,	Ala-230 to Tyr-238,	Thr-244 to Gln-255,	Pro-274 to Ser-279,	Thr-284 to Phe-306,	Leu-345 to Thr-354.	Thr-48 to Arg-56,
AA SEQ ID NO: Y		921	1171	922					_	7/11						1173			923									1174
ORF O: (From-To)		252 - 410	252 - 413	132 - 1550					2131 00	/101 - 66						1 - 534			30 - 1109									30 - 626
SEQ ID NO: X		306	556	307					633	/66						558			308									559
Contig ID:		871221	706332	877666					730504	/30204						470546			910133									904040
cDNA Clone ID		HRACD15	HRACD15	HRACJ35					110 4 CI16	HKACISS						HRACJ35			HRGBL78									HRGBL78
Gene No:		296		297															298									

	OMIM Disease	Reference(s):				7, 602080																							
						174810, 601567, 602080																							
	Cytologic Band					18q21.32														L									
	Predicted Epitopes		Pro-122 to Glu-127, Ala-136 to Tyr-141.		Pro-24 to Arg-32.	Ile-4 to Tyr-10,	Arg-119 to Pro-126,	Glu-152 to Gly-158,	Thr-209 to Phe-215.	Arg-40 to Pro-47,	Glu-73 to Gly-79,	Thr-130 to Phe-136,	Lys-277 to Lys-283.	Arg-40 to Pro-47,	Glu-73 to Gly-79,	Thr-130 to Phe-136.	Thr-19 to Thr-25.		Ser-58 to His-64.	Leu-51 to Gly-77,	Ile-117 to Pro-125.	Thr-25 to Cys-30,	Pro-35 to Arg-42.	Ile-46 to Tyr-56.	Tyr-24 to His-32,	Pro-38 to Ala-44,	Pro-66 to Glu-75,	His-111 to Gly-116,	1 yr-137 to 3c1-140,
AA	SEQ ID	NO: Y		1175	1176	924				1177				1178			925	926	927	928		1179		929	930				
	ORF (From-To)	X		11 - 19	1048 - 1146	10 - 1146				31 - 879				247 - 1104			122 - 268	155 - 328	104 - 406	142 - 570		122 - 256		98 - 271	299 - 1087	•			
	SEQ ID NO:	×		560	561	309				562				563			310	311	312	313		564		314	315				
	Contig ID:			904621	863802	1181699				1114849				1027712			827306	456536	545459	460527		371416		580872	692358				
	cDNA Clone ID			HRGBL78	HRGBL78	HROAJ39				HROAJ39				HROAJ39			HROBD68	HSAVD46	HSAVH65	HSAWD74		HSAWD74		HSAWZ41	HSDAJ46				
	Gene No:					299											300	301	302	303				304	305				

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OMIM Disease Reference(s):		Xq12-q13.3 300011, 300011, 300011, 300127, 305450,	309605, 313700, 313700, 313700, 313700,	313700, 314580																								
Cytologic Band		Xq12-q13.3																_		•								
Predicted Epitopes	Thr-176 to Ser-181, Lys-239 to Lys-249.	Val-29 to Val-37,	Asp-71 to His-76,	Gln-78 to Gly-84,	Met-105 to His-110,	Trp-117 to Asn-123,	Lys-179 to Pro-187,	Gly-218 to Asp-224,	Leu-237 to Ala-243,	Thr-256 to Asp-268,	Ser-275 to Lys-280,	Arg-308 to Glu-314,	Glu-326 to Glu-332,	Cys-343 to Asp-359.	Val-29 to Val-37,	Asp-71 to His-76,	Gln-78 to Gly-84,	Met-105 to His-110,	Trp-117 to Gly-122,	Gln-136 to Lys-141,	Leu-143 to Ala-149,	Thr-162 to Asp-174,	Ser-181 to Lys-186,	Arg-214 to Glu-220,	Glu-232 to Glu-238,	Cys-249 to Asp-265.	Phe-8 to Ser-13,	Val-81 to Arg-8/,
AA SEQ ID NO: Y		931													1180												932	
SEQ ORF ID NO: (From-To)		60 - 1256													126 - 1043					-							58 - 423	
SEQ ID NO: X		316													565												317	
Contig ID:		1352253													625998												1352287	
cDNA Clone ID		HSDEK49									•	•			HSDEK49												HSDEZ20	
Gene No:		306																									307	

ОМІМ	Disease Reference(s):																											
Cytologic	Band																5				•							
Predicted Epitopes		Asp-98 to Pro-104.	Phe-8 to Ser-13,	Ala-84 to Ser-90.	Ala-21 to Glu-31,	Thr-37 to Cys-43,	Asp-62 to Ser-79,	Lys-134 to Gly-146,	Leu-164 to Met-169,	Glu-171 to Lys-201.	Ala-21 to Glu-31,	Thr-37 to Cys-43,	Pro-64 to Asp-69.		Thr-32 to Lys-40,	Lys-146 to Glu-152.	His-170 to Pro-181,	Ser-204 to Pro-210.	His-172 to Pro-183, Ser-206 to Pro-212.		Glu-33 to Glu-56,	Thr-75 to Cys-81.	Glu-33 to Glu-56,	Thr-75 to Cys-81.	Tyr-15 to Leu-59,	Ala-68 to Asp-85,	Pro-87 to Asn-96,	HIS-120 to Lys-129,
AA SEQ	NO: Y		1181		933						1182			934	935		936		1183	1184	937		1185		938			
ORF	6		66 - 359		<i>191 - 66</i>						99 - 317			118 - 330	247 - 705		84 - 737		27 - 686	78 - 737	16 - 423		22 - 387		160 - 705			
SEQ	ID NO: X		995		318						267			319	320		321		895	695	322		270		323			
Contig			704101	- 	834619						836071			589974	795252		1036471		904821	905623	1301498		463645		545057			
cDNA Clone	Œ		HSDEZ20		HSDFJ26						HSDF126			HSDFW45	HSDJA15		HSDJL42		HSDJL42	HSDJL42	HSDSB09		HSDSB09		HSDSE75			
Gene	.ö.				308				_					309	310		311				312				313			

OMIM Disease Reference(s):			600631																	100690, 142989, 156232, 178600, 600258								
Cytologic Band			13q13.3																	2q31.1	-							
Predicted Epitopes	Ser-153 to Gln-170.	Leu-3 to Asn-9.	Asn-22 to Ile-29,	Glu-41 to Lys-50,	Arg-58 to Gln-73,	Gln-78 to Glu-89,	Val-91 to Glu-101,	Gln-109 to Arg-128,	Glu-133 to Thr-139,	Leu-146 to Cys-156,	Pro-163 to Trp-168,	Tyr-174 to Glu-198,	Leu-202 to Lys-213,	Gln-216 to Asn-223,	Leu-230 to Gly-238,	Gln-241 to Trp-246.	Asn-22 to Ile-29,	Ala-33 to Arg-51.	Glu-37 to Gly-45.	Pro-222 to Asn-231,	Asn-238 to Gly-247,	Ala-251 to Leu-264,	Ala-280 to Thr-285.	Pro-107 to Asn-116,	Asn-123 to Gly-132,	Ala-136 to Asn-150.	Gly-31 to Arg-36,	Thr-55 to Glu-62,
AA SEQ ID NO: Y		626	940												•		1186		941	942				1187			943	
SEQ ORF ID NO: (From-To)		44 - 73	117 - 884														150 - 326		8 - 184	49 - 906				393 - 908	•		786 - 3635	
SEQ ID NO: X		324	325														571		326	327				572			328	
Contig ID:		552789	1352248														612877		589447	702021				413210			1352409	
cDNA Clone ID		HSFAM31	HSICV24														HSICV24		HSIDJ81	HSKCP69				HSKCP69			HSKDA27	
Gene No:		314	315																316	317							318	

OMIM Disease Reference(s):		
Cytologic Band		
Predicted Epitopes	Ser-64 to Ser-79, Arg-87 to Asp-96, Arg-103 to Ala-109, Asp-120 to Arg-126, Gly-294 to Gly-302, Ser-305 to Ala-318, Val-320 to Arg-327, Pro-344 to Thr-351, Thr-383 to Thr-399, Leu-414 to Lys-435, Thr-449 to Ala-457, Gly-461 to Asn-479, Gly-461 to Asn-479, Gly-463 to Gln-498, Ser-503 to Arg-514, Lys-532 to Ala-559, Leu-563 to Ser-611, Lys-632 to Tyr-638, Asn-667 to Lys-672, Leu-701 to Met-707, Ser-745 to Lys-755, Lys-761 to Leu-768, Pro-787 to Trp-792, Lys-761 to Tyr-923, Ser-925 to Arg-939, Glu-942 to Tyr-950.	Gly-31 to Arg-36, Thr-55 to Glu-62, Ser-64 to Ser-79,
AA SEQ ID NO: Y		1188
SEQ ORF (From-To)		127 - 1653
SEQ ID NO:		573
Contig ID:		1074734
cDNA Clone ID		HSKDA27
Gene No:		

OMIM Disease Reference(s):																											
Cytologic Band																			•								
Predicted Epitopes	Arg-87 to Asp-96, Arg-103 to Ala-109,	Asp-120 to Arg-126,	Gly-294 to Gly-302, Ser-305 to Ala-318,	Val-320 to Arg-327,	Pro-342 to Thr-351,	Thr-383 to Thr-399,	Leu-414 to Lys-435,	Thr-449 to Ala-457,	Gly-461 to Asn-479,	Gly-483 to Gln-498,	Asn-504 to Val-509.	Gly-27 to Arg-32,	Thr-51 to Glu-58,	Ser-60 to Ser-75,	Arg-83 to Asp-92,	Arg-99 to Ala-105,	Asp-116 to Arg-122,	Gly-290 to Ala-314,	Val-316 to Arg-323,	Pro-338 to Arg-345,	Thr-358 to His-375,	Arg-403 to Ser-408,	Ser-420 to Ser-436,	Thr-447 to Ala-455,	Gly-459 to Asn-477,	Gly-481 to Gln-496,	Ser-501 to Arg-512,
AA SEQ ID NO: Y												1189															
SEQ ORF ID NO: (From-To) X												12 - 1673															
SEQ ID NO: X												574															
Contig ID:												872570															
Gene CDNA Clone No: ID												HSKDA27															
Gene No:																											

Market	OMIM Disease Reference(s):		188826																								
	Cytologic Band		22q13.2	1														·				-	15				
	Fredicted Epitopes	Lys-530 to Lys-554.	Ile-60 to Asn-69,	Leu-106 to Asp-112,	Glu-130 to Gly-136,	Phe-160 to Glu-167,	Pro-184 to Cys-190,	Glu-197 to Ser-202,	Arg-215 to Glu-221,	Thr-237 to Pro-242.	Thr-11 to Pro-22.	Gly-76 to Leu-83,	Ala-108 to Glu-113,	Ala-126 to Lys-132,	Gly-145 to Leu-151,	Gln-161 to Val-166,	Ala-180 to Gln-185,	Gly-190 to Ala-198,	Asn-203 to Gly-216.	Gly-76 to Leu-83,	Ala-108 to Glu-113,	Ala-126 to Lys-132,	יונכן מסד או כדו-נוט				Glu-23 to Asn-31, Thr-38 to Gly-48.
AA	SEQ ID NO: Y		944								1190	945								1611			946	1192	1193	947	948
	SEQ OKF ID NO: (From-To) X		353 - 1132								537 - 608	64 - 807								27 - 800			114 - 242	206 - 334	1331 - 1351	220 - 327	225 - 389
9	SEQ ID NO: X		329								575	330								925			331	577	578	332	333
	Config ID:		676075								409905	1307105								552233			1016920	852244	895206	467397	1352201
7	Gene CDNA Clone No: ID		HSKGN81								HSKGN81	HSKHZ81								HSKHZ81			HSI 1G37	HSI 1G37	HSLJG37	HSNAD72	HSNMC45
	Sene No:		319									320											321			322	323

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OMIM Disease	Reference(s):		230400, 250250	100690, 142989, 156232, 178600, 600258																					182600, 186880, 190195, 190195, 222700,	600243, 602279, 602279		
Cytologic Band			9p13-p12	2q31.3																					14q11.2			
Predicted Epitopes			Pro-22 to Lys-29.	Ala-19 to Val-31,	Arg-38 to Gly-49,	Ala-61 to Lys-66,	Tyr-68 to Pro-78,	Gly-116 to Ala-121,	Asp-154 to Ser-162,	Glu-173 to Gln-186,	Phe-194 to Gly-203,	Pro-207 to Val-212.	Ala-19 to Val-31,	Arg-38 to Gly-49,	Ala-61 to Lys-66,	Tyr-68 to Pro-78,	Gly-116 to Ala-121,	Asp-154 to Ser-162,	Glu-173 to Gln-186,	Phe-194 to Gly-203, Pro-207 to Val-212	Ala-19 to Val-31,	Arg-38 to Asp-50.	Ser-6 to Arg-15.		Pro-7 to Cys-12,	Lys-48 to Tyr-62,	Arg-182 to His-187,	Leu-107 to Ott-170,
AA SEQ ID	NO: Y	1194	949	950									1195								1196		951	952	953			
ORF (From-To)	X	232 - 309	130 - 318	87 - 743									91 - 747								86 - 256		96 - 332	82 - 207	344 - 2161			
SEQ ID NO:	X	579	334	335									580								581		336	337	338			
Contig ID:		545060	1658E9	1306702									602258								401251		460537	892171	1352343			
Gene cDNA Clone No: ID		HSNIMC45	HSQCM10	HSQEO84									HSQE084								HSOE084	,	HSQFP66	HSRFZ57	HSSGD52			
Gene No:			324	325																			326	327	328			

OMIM Disease Reference(s):				116860, 129900, 233700, 600079	278700, 602088 141750, 141800, 141800, 141800, 141800, 141850, 141850, 141850, 141850, 156850, 186580, 191092, 600140, 600273, 601313, 601785
Cytologic Band				7q11.23	9q22.31 16p13.3
Predicted Epitopes	Thr-211 to Gly-226, Leu-270 to Thr-275, Gly-278 to Gly-289, Pro-444 to Asn-449, Glu-453 to Lys-461, Gly-491 to Thr-496, Ser-525 to Trp-532.	Pro-7 to Cys-12, Lys-48 to Tyr-62, Arg-182 to His-187, Leu-189 to Glu-196, Thr-211 to Gly-226, Leu-270 to Thr-275, Gly-278 to Gly-289, Pro-444 to Asn-449, Glu-453 to Lys-461, Gly-491 to Thr-496, Ser-525 to Trp-532.	Asp-23 to Gly-29. Met-33 to Pro-39, Ser-74 to Trp-79.	Asp-26 to Asn-31, Ser-37 to His-49, Ala-65 to Ser-73.	Ser-47 to Pro-57, Ser-77 to Glu-82, Thr-90 to Trp-98, Arg-124 to Lys-137,
AA SEQ ID NO: Y		1197	954	926	958
SEQ ORF ID NO: (From-To)		338 - 2155	153 - 323 63 - 329	256 - 528	295 - 432 47 - 964
SEQ ID NO: X		582	339	341	343
Contig ID:		845666	413246	898965	634032
cDNA Clone ID		HSSGD52	HSUBW09 HSVAT68	HSVBU91	HSYBG37
Gene No:			329	331	332

OMIM Disease Reference(s):			225500, 600593, 602363	
Cytologic Band			4p16-p15	
Predicted Epitopes	Ala-183 to Glu-192, Lys-220 to Gln-229, Asn-244 to Arg-258, Thr-271 to Asn-278, Glu-285 to Gly-297.	Ser-47 to Pro-57, Ser-77 to Glu-82, Thr-90 to Trp-98, Arg-124 to Lys-137, Ala-183 to Glu-192, Lys-220 to Gln-229, Asn-244 to Arg-258, Thr-271 to Asn-278, Glu-285 to Glu-297	Gly-16 to Pro-30, Pro-42 to Gly-56, Gly-62 to Gly-77, Glu-93 to Gly-104, Glu-109 to Glu-114, Pro-121 to Gly-134, Ser-157 to Arg-162, Glu-174 to Thr-182, He-283 to Leu-289.	Gly-16 to Pro-30, Pro-42 to Gly-56, Gly-62 to Gly-77, Glu-93 to Gly-104, Glu-109 to Glu-114, Pro-121 to Asp-126.
AA SEQ ID NO: Y		1198	959	1199
ORF O: (From-To)		48 - 965	106 - 972	107 - 490
SEQ ID NO:		583	344	584
Contig ID:		581098	1352172	456551
cDNA Clone ID		HSYBG37	HSZAF47	HSZAF47
Gene No:			334	

OMIM Disease Reference(s):	107300, 131210, 136132, 145001, 173610, 601652																					
Cytologic Band	1923.1														•	•				21921.2		
Predicted Epitopes	Glu-15 to Arg-23, Asn-79 to Gly-84, Ser-101 to Gly-106, Ser-111 to Asn-116.	1200 Glu-15 to Arg-23, Asn-79 to Gly-84.	Pro-255 to Leu-264.			Gly 41 to Leu-46,	Asp-67 to Thr-75,	lle-114 to Gly-122, Pro-156 to Tro-161.	Gly-41 to Leu-46,	Asp-67 to Thr-75,	Glv41 to I.en-46.	Asp-67 to Thr-75,	Ile-114 to Ala-123.	Glu-43 to Asn-49,	Cys-75 to Lys-88,	Glu-120 to Asp-125,	Pro-182 to Ser-188,	Pro-210 to Gln-216.	Glu-43 to Asn-49.	Met-1 to His-7.	Gly-35 to Gly-40.	Asp-61 to Gln-68,
AA SEQ ID NO: Y	096	1200	961	1201	1202	962			1203		1204			963					1205	964	965	996
ORF O: (From-To)	92 - 520	84 - 512	319 - 1167	372 - 737	124 - 771	13 - 546			21 - 404		27-518	i		19 - 717					19 - 252	59 - 952	231 - 371	26 - 799
SEQ ID NO: X	345	585	346	586	587	347			588		589		,	348					590	349	350	351
Contig ID:	753289	457172	1018291	882919	864120	1352365			877448		666743			1352193					519372	206980	543396	908143
cDNA Clone ID	HTADX17	HTADX17	HTAEE28	HTAEE28	HTAEE28	HTECC05			HTECC05		HTECC05			HTEDY42					HTEDY42	HTEEB42	HTEFU65	HTEGI42
Gene No:	335		336			337								338						339	340	341

OMIM Disease Reference(s):						139190, 139190, 224100, 601002, 601002,	601146, 601146, 601146								164500, 176880, 232500, 600151, 600795					231680, 276700						
Cytologic Band						20q11.2			20pter-q11.23						3p12-p11.1			18		15q25		11,13				
Predicted Epitopes	Gly-180 to Lys-185.			Pro-1 to Arg-15.		Leu-67 to Glu-73,	Arg-83 to Gln-92,	Leu-124 to Tyr-134, Gln-146 to Thr-157.	Arg-21 to Thr-29,	Tyr-56 to Lys-63,	Ser-93 to Ser-100,	Glu-109 to Lys-116.	Arg-21 to Thr-29.	Glu-33 to Arg-45.		Pro-98 to Gln-106.	Arg-71 to Ala-82.			Gly-85 to Lys-94,	Gln-125 to Cys-131, Glu-151 to Gly-159.	Gly-10 to Gly-17,	Pro-49 to Glu-54,	Gln-97 to Asp-103,	Ser-120 to Tyr-125,	Gln-186 to Leu-199,
AA SEQ ID NO: Y		1206	1207	1208	1209	<i>L</i> 96			896				1210	696	970	971	972	973	1211	974		975				
ORF O: (From-To)		145 - 915	1 - 282	1081 - 1326	670 - 849	121 - 1059			188 - 616				187 - 528	22 - 198	164 - 298	15 - 491	365 - 634	231 - 347	224 - 340	527 - 1069		30 - 2495				
SEQ ID NO: X		591	592	593	594	352			353				595	354	355	356	357	358	965	359		360				
Contig ID:		904624	850770	847564	830165	600394			722254				423009	520468	836072	847090	834931	908144	906536	706618		1040047				
cDNA Clone ID		HTEGI42	HTEGI42	HTEGI42	HTEGI42	HTEHU31			нтен093				HTEHU93	HTEIP36	HTELP17	HTELS08	HTEPG70	HTHCA18	HTHCA18	HTJMA95		HTJML75				
Gene No:						342			343					344	345	346	347	348		349		350		_		

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OMIM Disease Reference(s):			103850, 114835, 116800, 140100, 140100, 192090, 192090, 192090, 192090, 245900, 276600, 600223		250790	
Cytologic Band			16q22.1		18q23	
Predicted Epitopes	Glu-202 to Tyr-213, Ser-225 to Cys-233, Thr-269 to Ser-284, Gly-308 to Val-328, Asg-350 to Ala-357, Arg-367 to Gln-372, Arg-429 to Thr-449, Gly-444 to Thr-449, Thr-466 to Val-481, Val-485 to Ser-499, Ser-534 to Arg-540, Met-564 to Ile-570, Asn-573 to Phe-589, Pro-603 to Val-611, Arg-706 to Gly-711, Glu-717 to Asp-725, Ser-732 to Ser-738, Gln-743 to Glu-749, Leu-799 to Asp-805.	Gly-49 to His-56.	Ser-36 to Trp-41, Pro-53 to Arg-58.	Ser-33 to Lys-43.	Asp-32 to Glu-37, Ala-41 to Phe-46, His-171 to Ala-176.	Ala-23 to His-34, His-153 to Ala-158.
AA SEQ ID NO: Y		1212	976	776	978	1213
SEQ ORF ID NO: (From-To)		335 - 529	33 - 248	73 - 378	124 - 687	189 - 698
SEQ ID NO: X		265	361	362	363	298
Contig ID:		873355	519329	634852	1352310	791409
Gene cDNA Clone No: ID		HTJML75	HTLAA40	HTLEP53	HTLFES7	HTLFE57
Gene No:			351	352	353	

OMIM Disease Reference(s):																									
Cytologic Band		3	٠	20q13.33							11						-					_	-		
Predicted Epitopes	Ala-23 to His-34, His-153 to Ala-158.			Gln-27 to Arg-36.	Gly-33 to Arg-40,	Ser-106 to Met-112, Ala-154 to Gly-163.			Arg-1 to Gly-7, Phe-11 to Arg-23.		Leu-21 to Ala-30,	Ser-38 to Asp-47,	Pro-87 to Asp-94,	Leu-197 to Thr-204,	Pro-256 to Ser-262,	Thr-277 to Arg-282,	Thr-293 to Val-302,	Lys-315 to Arg-321.	Leu-21 to Ala-30,	Ser-38 to Asp-47,	Pro-87 to Asp-94,	Leu-197 to Thr-204,	Pro-256 to Ser-262,	Thr-277 to Arg-282,	1 nr-293 to 1 rp-303.
AA SEQ ID NO: Y	1214	626	086	186	982		983	1215	1216	1217	984								1218						
SEQ ORF ID NO: (From-To)	110-619	110 - 364	7 - 129	43 - 222	155 - 727		30 - 215	23 - 208	71 - 1036	1555 - 1596	178 - 1263								302 - 1390						
SEQ ID NO:	599	364	365	366	367		368	009	601	602	1								603						
Contig ID:	608317	1046341	519313	526021	604983		1028538	848199	848200	848196	1317835								581435						
Gene CDNA Clone No: ID	HTLFE57	HTLIV19	HTNB091	HTODK73	HTOHD42		HTOHM15	HTOHM15	HTOHM15	HTOHM15	HTPBW79								HTPBW79						
Gene No:		354	355	356	357		358				359														

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	OMIM . Disease	Reference(s):								107300, 131210, 136132, 145001, 173610,	601652		300046, 300067, 300067, 300121, 300121,	301201, 301835, 311850												1p36.13-q41 115665, 120550, 120570, 120575, 130500,	133200, 167410, 172430, 600975			
	Cytologic Band									1923.1			Xq22.3-23													1p36.13-q41				
	Predicted Epitopes		Leu-21 to Ala-30,	Ser-38 to Asp-47,	Pro-87 to Asp-94,	Leu-197 to Are-202	Pro-287 to Ser-293.	TL- 200 42 A 22 212	Inr-306 to Arg-313, Thr-324 to Tro-334.				Ser-29 to Ser-34,	Ser-186 to Asp-196,	Arg-206 to Ser-225.	Ser-29 to Ser-34.			Glu-55 to Arg-61,	Gln-84 to Ser-92,	Ser-99 to Ser-104.	Leu-37 to Asn-42.	Leu-37 to Asn-42.	Lys-41 to Arg-46.	Thr-54 to Ile-59.	Glu-24 to Tyr-35,	Arg-83 to Thr-92,	Pro-148 to Gly-154.	Asp-51 to His-56.	Ala-45 to Gly-50.
AA	SEQ	NO: Y	1219				·			985		1220	986			1221	1222	286	886			686	1223	1224	066	166				993
	SEQ ORF ID NO: (From-To)		92 - 1336							2365 - 2577		530 - 745	118 - 810			111 - 530	96 - 353	170 - 283	133 - 534			95 - 223	100 - 228	175 - 402	334 - 639	217 - 822			328 - 498	72 - 230
	SEQ ID NO:	X	604							370		509	371			909	<i>L</i> 09	312	373			374	809	609	375	376			211	378
	Contig ID:		396459							854941		266683	916616			895024	880868	460579	637725			1008159	863187	754125	429618	695765			603918	838288
	cDNA Clone ID		HTPBW79							HTPCS72		HTPCS72	нтрін83			нтрін83	HTPIH83	HTSEW17	HTTBI76			HTTBS64	HTTBS64	HTTBS64	HTWCT03	HTXDW56			HTXJM03	HTXON32
	Gene No:									360			361					362	363			364			365	366			367	368

OMIM	Disease Reference(s):																												
Cytologic	Band	20q11.23																				-							
Predicted Epitopes		Pro-24 to Pro-37.	Pro-24 to Pro-37.	Ser-44 to Leu-51,	Arg-81 to Cys-94,	Thr-132 to Tyr-140,	Arg-143 to Ile-154.	Ser-44 to Leu-51,	Arg-81 to Cys-94,	Thr-118 to Tyr-126,	Arg-129 to Ile-140.	Pro-31 to Ala-37.	Trp-35 to Trp-45,	Pro-52 to Asp-57,	Thr-73 to Arg-82,	Pro-105 to Leu-112,	Pro-115 to Arg-127,	Pro-140 to Gln-151.	Trp-35 to Trp-45,	Pro-52 to Asp-57,	Thr-73 to Thr-80,	Pro-96 to Leu-103,	Pro-106 to Arg-118,	Pro-131 to Gln-142.	Trp-35 to Trp-45,	Pro-52 to Asp-57,	Thr-73 to Thr-80,	Pro-96 to Leu-103,	Pro-106 to Leu-119.
AA SEO		994	1225	995				1226				966	266						1227						1228				
ORF	ID NO: (From-To)	1085 - 1303	197 - 361	49 - 525				74 - 508				123 - 275	286 - 738	•		•			144 - 572						55 - 414				
SEO	ID NO: X	379	019	380				611				381	382						612						613				
Contig	ë	1352211	562791	1352349				846380				638402	1352424						1300737						603538				
cDNA Clone	Œ	HUDBZ89	HUDBZ89	HUFBY15				HUFBY15				HUFCJ30	HUKAHSI						HUKAHSI						HUKAH51				
Gene	 2	369		370								371	372																

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OMIM Disease	Reference(s):	106150, 106150, 145260, 173870, 173870,	000/27, 000270, 001/44, 0012/3																									112261, 176640, 176640, 176640, 236700, 601920	
Cytologic Band		1942																				-						20p12	2
Predicted Epitopes		Thr-35 to Lys-43,	F10-39 to A1g-04.	Gly-39 to Thr-44,	Asn-51 to Thr-62,	Pro-88 to Pro-104,	Ser-109 to Phe-124,	Ala-190 to Asn-196,	Gln-388 to Glu-394,	Gln-402 to Gly-409,	Asn-427 to Leu-439,	Glu-447 to Thr-453,	Pro-468 to Gln-474,	Pro-476 to Phe-482,	Arg-498 to Arg-504,	Arg-508 to Arg-518.	Gly-39 to Thr-44,	Asn-51 to Thr-62,	Pro-88 to Pro-104,	Ser-109 to Ser-114.	Gln-54 to Gly-61,	Asn-79 to Leu-91,	Glu-99 to Thr-105,	Pro-120 to Gln-126,	Pro-128 to Phe-134,	Arg-150 to Arg-156,	Arg-160 to Arg-170.		Arg-128 to Tyr-134.
SEQ ID	Z	866	٦	666													1229				1230			•				0001	1001
SEQ ORF ID NO: (From-To)		74 - 1594		280 - 1845										·			281 - 1666				179 - 703							14 - 151	111 - 668
SEQ ID NO:	X	383		384													614			•	615							385	386
Contig ID:		694590		1352367													883176				655372							571200	1194812
cDNA Clone		HUKBT29		HUSXS50													HUSXS50				HUSXS50							HUVEB53	HVARW53
Gene No:		373		374								_																375	376

						200,		260,	518,																		
OMIM Disease Reference(s):						120550, 120570, 120575, 130500, 133200, 600975		107300, 131210, 136132, 145001, 145260,	173610, 276901, 600332, 600759, 601518,	., 601744, 601975																	
						120550, 600975	600320	107300	173610	601652												602629					
Cytologic Band		,				1p36.31- p36.11	6q24.3	1q24-q41														8p21.3					
Predicted Epitopes		Pro-53 to Trp-61.			Pro-30 to Asn-36.			Gln-20 to Phe-25,	Gly-58 to Ala-66,	Gln-69 to Leu-74,	Asn-8/ to Ile-100,	Thr-135 to Trp-142.	Gln-20 to Phe-25,	Gly-58 to Ala-66,	Gln-69 to Leu-74,	Asn-87 to Ile-100,	Thr-135 to Trp-142.	Trp-47 to Thr-54,	Ser-68 to Asn-73,	Ser-86 to Gly-92.	Trp-47 to Thr-54.	Ser-25 to Phe-31.	Ser-25 to Phe-31,	Lys-55 to Arg-61.		Pro-16 to Phe-21,	Pro-24 to Arg-35,
AA SEQ ID NO: Y	1231	1002	1232	1233	1003	1004	1005	1006					1234	•		·		1007	•		1235	1008	1236		1009	1010	
SEQ ORF ID NO: (From-To)	96 - 590	322 - 825	322 - 483	312 - 818	57 - 203	581 - 709	156 - 383	37 - 600			•		35 - 598			-		243 - 560			233 - 550	1342 - 1542	132 - 314		271 - 426	131 - 694	
SEQ ID NO: X	616	387	617	618	388	389	390	391					619					392			620	393	621		394	395	
Contig ID:	1044491	838626	680888	793875	580889	799506	836469	1093347					886210					846382			646977	1352265	98238		799427	805642	
cDNA Clone ID	HVARW53	HWAAD63	HWAAD63	HWAAD63	HWABA81	HWADJ89	HWBAR88	HWBCB89					HWBCB89					HWBCP79			HWBCP79	HWBDP28	HWBDP28		HWBFX31	HWHHL34	
Gene No:		377			378	379	380	381										382				383			384	385	

	OMIM	Disease Reference(s):																													
	Cytologic	Band																													
	Predicted Epitopes		Arg-92 to Pro-98,	Asn-143 to Lys-151,	Leu-169 to Ile-176.	Arg-40 to Pro-46.	Pro-16 to Phe-21,	Pro-24 to Arg-35,	Arg-92 to Pro-98,	Asn-143 to Lys-151,	Leu-169 to Ile-176.	Val-35 to Lys-41,	Ser-68 to Gln-73,	Glu-88 to Glu-93,	Arg-156 to Gly-163,	Ala-199 to Gly-206,	Asp-216 to Ser-226,	Thr-249 to Asn-254,	Asp-339 to Pro-345,	Ile-370 to Gly-379,	Pro-429 to Glu-434,	Arg-461 to Pro-466,	Ala-475 to Thr-482,	Pro-585 to Gly-593,	Glu-631 to Gln-639,	Pro-674 to Pro-682,	Gln-715 to Gly-720,	Ser-736 to Arg-742.		Gly-31 to Thr-51.	
AA	SEQ	NO: Y				1237	1238					1011																	1012	1013	1014
	ORF	ID NO: (From-To)				209 - 517	101 - 664					169 - 2397									•								129 - 626	190 - 378	157 - 297
	SEQ	ID NO: X				622	623					396																	268	398	399
	Contig	Ä				801943	341560					762842																	793713	826754	610383
	Gene cDNA Clone	a				HWHHL34	HWHHL34					HWHQS55																	HWLIH65	HYAAJ71	HYBAR01
	Gene	ë Ž										386																	387	388	389

OMIM	Reference(s):																												
Cytologic Band																									-				
Predicted Epitopes		Pro-34 to Trp-41.	Leu-3 to Arg-8,	Asp-57 to Arg-64,	Glu-66 to Thr-75,	Arg-120 to Ile-126,	Gln-161 to Asp-177,	Thr-182 to Ser-194,	Lys-211 to Gln-216,	Asn-274 to Gly-290,	Thr-296 to Phe-302.	Leu-3 to Arg-8,	Asp-57 to Arg-64,	Glu-66 to Thr-75,	Arg-120 to Ile-126,	Gln-161 to Asp-177,	Thr-182 to Ser-194,	Lys-211 to Gln-216,	Asn-274 to Gly-290,	Thr-296 to Phe-302.	Leu-3 to Arg-8,	Asp-57 to Arg-64,	Glu-66 to Thr-75,	Arg-120 to Ile-126,	Gln-161 to Asp-177,	Thr-182 to Ser-194,	Lys-211 to Gln-216,	Asn-274 to Gly-290,	Thr-296 to Phe-302.
AA SEQ	NO: Y	1015	1016									1239									1240								
ORF (From.To)	(2.1.01.1)	319 - 444	468 - 1400	** *	******							468 - 1400				-					468 - 1400				-		-	-	
SEQ.	X	400	401									624									625								
Contig		834784										887467								:	878627			. — 					
Gene cDNA Clone		HYBBE75	HAPSA79									HAPSA79									HAPSA79								
Gene No.		390	391																										

'ABLE 1B.2

			CED	
Gene	Gene cDNA Clone Contig ID:	Contig ID:	A C	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
-	H2CBU83	884134	=	AR182:8, AR314:7, AR271:7, AR280:6, AR270:5, AR165:5, AR162:5,
				AR039:5, AR192:5, AR163:4, AR193:4, AR207:4, AR176:4, AR269:4, AR175:4, AR226:4, AR243:4, AR217:4,
				AR273:4, AR168:4, AR282:4, AR204:4, AR291:4, AR265:4, AR183:4, AR274:4, AR299:4, AR214:4, AR205:4,
				AR206:4, AR194:4, AR060:4, AR272:4, AR238:4, AR186:4, AR222:4, AR053:4, AR197:4, AR089:3, AR257:3,
		· 		AKL93:3, AKL89:3, AK411:3, AK221:3, AK171:3, AK191:3, AK230:3, AK232:3, AK272:3, AK273:3, AK309:3, AK309:3, AK177:3 AR173:3 AR
				AR201:3, AR285:3, AR189:3, AR296:3, AR181:3, AR300:3, AR185:3, AR253:3, AR202:3, AR281:3, AR237:3,
				AR184:3, AR268:3, AR233:3, AR286:3, AR232:3, AR308:3, AR277:3, AR267:3, AR228:3, AR288:3, AR316:3,
				AR239:3, AR195:2, AR242:2, AR263:2, AR033:2, AR287:2, AR196:2, AR210:2, AR259:2, AR174:2, AR294:2,
				AR096:2, AR234:2, AR293:2, AR290:2, AR190:2, AR255:2, AR055:2, AR213:2, AR264:2, AR231:2, AR313:2,
				AR297:2, AR258:2, AR170:2, AR218:2, AR247:2, AR061:2, AR236:2, AR219:2, AR198:2, AR230:2, AR254:2,
				AR256:2, AR261:2, AR104:2, AR240:2, AR262:2, AR283:2, AR229:2, AR227:2, AR260:2, AR200:1, AR203:1,
				AR179:1, AR244:1, AR199:1 S0414:9, S0422:7, L0662:7, S0444:6, L0748:4, L0581:4, S0442:3, H0031:3, L0666:3,
				L0754:3, H0656:2, S0358:2, S0360:2, H0013:2, S0438:2, S0440:2, L0598:2, L0803:2, L0540:2, L0756:2, L0752:2,
				L0758:2, L0759:2, S0242:2, H0624:1, S0282:1, H0742:1, H0393:1, H0586:1, H0574:1, H0036:1, H0004:1, T0103:1,
				T0110:1, H0571:1, H0569:1, H0123:1, L0471:1, H0594:1, S6028:1, H0622:1, UNKWN:1, L0649:1, L0381:1,
				L0776:1, L0659:1, L0528:1, L0792:1, L0793:1, L0663:1, L0664:1, L0665:1, L2257:1, H0144:1, S0374:1, H0547:1,
				H0593:1, H0690:1, H0670:1, H0648:1, H0672:1, H0651:1, H0539:1, S0378:1, S0380:1, H0521:1, S0406:1, H0555:1,
	H2CB1183	745366	402	104 (0:1, LO/44:1, LO/51:1 alid 502/0:1.
2	H2MAC30	544957		AR096:11, AR039:10, AR313:10, AR299:10, AR250:9, AR240:8, AR254:8, AR055:8, AR242:8, AR060:7, AR089:7.
			}	AR162:7, AR316:6, AR161:6, AR163:6, AR213:6, AR269:6, AR252:5, AR268:5, AR169:5, AR200:5, AR204:5,
				[AR215:5, AR165:5, AR053:5, AR196:5, AR166:5, AR164:5, AR199:5, AR104:5, AR282:5, AR176:5, AR266:5,
				AR180:4, AR264:4, AR261:4, AR277:4, AR300:4, AR229:4, AR183:4, AR181:4, AR190:4, AR173:4, AR263:4,
				AR247:4, AR309:4, AR197:4, AR274:4, AR178:4, AR214:4, AR205:4, AR212:4, AR243:4, AR312:4, AR191:4,
				AR255:4, AR182:4, AR236:4, AR170:4, AR245:3, AR185:3, AR272:3, AR217:3, AR171:3, AR267:3, AR175:3,
				AR308:3, AR192:3, AR290:3, AR271:3, AR193:3, AR291:3, AR219:3, AR237:3, AR233:3, AR188:3, AR201:3,
				AR216:3, AR311:3, AR270:3, AR177:3, AR174:3, AR218:3, AR234:3, AR283:3, AR179:3, AR293:3, AR207:3,
				AR231:3, AR221:3, AR228:3, AR203:3, AR285:3, AR265:3, AR255:2, AR224:2, AR288:2, AR238:2, AR195:2,

Gene	cDNA Clone	Contig ID	SEQ NO.X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
				AR287:2, AR257:2, AR239:2, AR168:2, AR297:1, AR222:1, AR232:1, AR33:1, AR258:1, AR210:1, AR226:1, AR210:1,
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6	HAGAI85	381942	19	AR282:65, AR104:12, AR218:12, AR240:12, AR096:11, AR219:11, AR316:8, AR185:8, AR060:7, AR299:7, AR055:6, AR300:4 AR089:4 AR075:4 AR089:3 AR083:3 AR083
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16	HAICP19	422672	26	AR277:26, AR283:23, AR219:15, AR089:14, AR282:14, AR185:14, AR240:14, AR218:13, AR316:13, AR104:12,
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17	HAJAN23	1352364	27	AR192:7, AR169:6, AR207:6, AR170:6, AR168:5, AR214:5, AR161:5, AR162:5, AR165:5, AR163:5, AR163:5,
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18	HAJBR69	638516	28	AR309:4, AR242:3, AR217:3, AR235:3, AR225:3, AR170:2, AR252:2, AR263:2, AR180:2, AR171:2, AR282:2,
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	HAMFC93	906819	411	
21	HAMFE15	\$69506	31	AR235:3, AR275:3, AR221:3, AR282:2, AR207:2, AR291:2, AR180:2, AR286:2, AR173:2, AR178:2, AR225:2, AR243:3 AR277:1 AR181:1 AR181:1 AR161:1 AR285:1 AR168:1 AR267:1 AR277:1 AR261:1
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				L0471:1, H0012:1, S0022:1, L0483:1, H0644:1, H0111:1, H0673:1, S0036:1, H0135:1, H0038:1, H0268:1, H0413:1,

Gene No:	cDNA Clone ID	SEQ ID Contig ID: NO:X	SEQ NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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				S0406:1, H0555:1, H0345:1, L0747:1, L0777:1, L0752:1, S0260:1, H0445:1, H0707:1, S0434:1, S0436:1, L0597:1, L0591:1, L0599:1, L0601:1, H0653:1, H0542:1, H0543:1, S0042:1 and S0424:1.
23	HAMGR28	892971	33	AR271:8, AR184:7, AR060:7, AR240:6, AR089:6, AR219:5, AR104:5, AR183:5, AR282:5, AR052:5, AR275:5,
				AR266:5, AR316:5, AR2/4:5, AR249:5, AR192:4, AR055:4, AR267:4, AR096:4, AR247:4, AR2/7:4, AR310:3, AR389:4, AR312:4, AR283:4, AR288:4, AR285:4, AR186:4, AR182:4, AR185:4, AR288:4, AR289:4, AR289:4, AR289:3,
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				L0764:1, L0767:1, L0649:1, L0803:1, L0806:1, L0653:1, L0659:1, L0518:1, L0789:1, L0791:1, S0053:1, H0144:1,
				H0701:1, H0725:1, S0148:1, L0438:1, H0519:1, H0593:1, S0406:1, L0748:1, L0745:1, L0749:1, L0750:1, L0779:1,
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24	HANGG89	845690	34	AR089:9, AR218:8, AR096:8, AR104:7, AR219:7, AR283:7, AR039:6, AR316:6, AR313:6, AR263:6, AR060:5,
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				AR297:2, AR174:2, AR257:2, AR243:2, AR288:2, AR189:2, AR216:2, AR214:2, AR269:2, AR283:2, AR170:2, AR262:2, AR191:2, AR191:2, AR192:2, AR195:2, AR195:2, AR195:2, AR195:2, AR195:2, AR195:2,
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				J.0/55:1.

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HANGG89	844216	415	
HANGG89	692291	416	
HAPOM49	769555	35	AR089:5, AR169:5, AR060:5, AR282:4, AR283:4, AR055:3, AR218:3, AR096:3, AR171:3, AR104:3, AR277:3,
			AR313:3, AR217:3, AR039:2, AR240:2, AR316:2, AR221:2, AR163:2, AR180:2, AR183:2, AR170:2, AR172:3, AR3172:3, AR316:3, AR306:3, AR
			AR10512, AR29912, AR10012, AR24212, AR19512, AR10012, AR50012, AR21512, AR10212, AR10411, AR21011, AR10411, AR19611, AR19611, AR19611, AR19611, AR19611, AR20511, AR20511, AR20611, AR2
			AR270:1, AR268:1, AR289:1, AR245:1, AR312:1, AR223:1, AR212:1, AR261:1, AR219:1, AR297:1, AR192:1
			S0406.5, L0750.5, L0777.4, L0749.3, L0779.3, H0662.2, S0440.2, L0770.2, L0794.2, L0776.2, L0657.2, L0783.2,
			L0740.2, L0747.2, L0780.2, S0420.1, S0442.1, S0444.1, S0045.1, L3316.1, H0599:1, H0575:1, S0474:1, T0115:1,
			H0083:1, H0510:1, H0644:1, H0551:1, S0386:1, H0494:1, H0561:1, H0538:1, S0422:1, L0646:1, L0804:1, L0774:1,
			L0809:1, L0530:1, L0663:1, L0664:1, L0665:1, H0593:1, S0380:1, S0027:1, L0748:1, L0439:1, L0750:1, L0753:1, L0758:1, L07
HAPOM40	722386	417	
LIA DEWING	1757770	٠,	AD174.74 AD235.73 AD106.73 AD177.77 AD101.10 AR175.10 AR233.10 AR728.10 AR179.18 AR190.17.
2	9/77661		AR203:17, AR257:17, AR178:17, AR182:17, AR188:17, AR060:17, AR176:17, AR181:16, AR295:16, AR261:16,
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			AR055:11, AR300:11, AR228:11, AR089:11, AR316:11, AR275:10, AR297:10, AR211:10, AR289:10, AR239:9,
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			AR192:1 L0748:12, S0474:5, L0777:5, L0758:5, H0424:4, H0038:4, L0752:4, L0774:3, L0742:3, L0779:3, L0755:3,
			H0616:2, L0770:2, L0764:2, L0776:2, H0539:2, L0753:2, L0599:2, H0663:1, H0722:1, H0728:1, H0208:1, S0045:1,
			L3388:1, L3484:1, L3491:1, T0040:1, H0575:1, S0010:1, S0049:1, H0052:1, H0545:1, H0009:1, H0103:1, H0012:1,
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			S0392:1, S0027:1, L0747:1, L0786:1, L0731:1, L0757:1, L0759:1, L0591:1 and H0653:1.

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Tissue Distribution Library Code:Count (see Table 4 for Library Codes)		AR196:22, AR161:20, AR162:20, AR163:19, AR207:17, AR214:17, AR223:16, AR165:15, AR164:14, AR222:14, AR235:14, AR168:14, AR245:13, AR264:13, AR166:13, AR242:13, AR200:13, AR172:12, AR217:12, AR288:12, AR170:12, AR195:12, AR224:12, AR264:12, AR261:12, AR225:11, AR211:11, AR172:11, AR171:11, AR192:11, AR244:11, AR221:11, AR176:11, AR207:10, AR210:10, AR193:10, AR211:10, AR236:10, AR246:9, AR197:9, AR198:9, AR216:9, AR216:9, AR311:9, AR243:9, AR189:8, AR272:8, AR308:8, AR174:8, AR246:9, AR197:9, AR212:8, AR295:6, AR240:8, AR216:1, AR265:7, AR265:5, AR266:5, AR266:3, AR266:1, A	AR055:34, AR104:32, AR283:31, AR089:31, AR219:23, AR096:22, AR218:22, AR060:21, AR313:20, AR316:15, AR185:14, AR039:13, AR299:12, AR182:10, AR28:10, AR294:10, AR267:9, AR240:7, AR257:7, AR293:6, AR233:6, AR300:5, AR164:5, AR258:5, AR221:5, AR260:5, AR170:5, AR288:5, AR277:5, AR175:4, AR285:4, AR233:6, AR262:4, AR255:4, AR258:4, AR280:3, AR260:3, AR260:3, AR260:3, AR260:3, AR260:3, AR260:3, AR260:3, AR270:3, AR170:3, AR163:3, AR165:2, AR261:2, AR260:2, AR260:2, AR270:2, AR290:2, AR270:3, AR270:3, AR173:2, AR250:2, AR290:2, AR271:2, AR290:2, AR271:2, AR290:2, AR271:2, AR290:2, AR271:2, AR290:2, AR271:2, AR290:2, AR271:2, AR290:2, AR271:3, AR290:2, AR271:3, AR290:1, AR190:1, AR188:1, AR271:1, AR193:1, AR271:1, AR271:1, AR272:1, AR272:		AR313:46, AR173:29, AR258:29, AR096:29, AR229:29, AR300:26, AR218:26, AR240:26, AR247:26, AR214:26, AR196:24, AR223:23, AR175:23, AR257:22, AR174:22, AR178:22, AR165:21, AR217:21, AR162:21, AR183:21, AR161:21, AR089:21, AR297:20, AR163:20, AR264:20, AR164:20, AR033:20, AR309:20, AR216:19, AR181:19,
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Contig ID:	684272	834358	1352276	92830	635514
cDNA Clone Contig ID: NO.	HAPPW30	HAPUC89	HATAC53	HATAC53	HATBR65
Gene No:	П	27	28		29

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Gene No:	cDNA Clone	Contig ID:	S B S	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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				AR060:13, AR291:13, AR222:13, AR260:13, AR200:12, AR104:12, AR211:12, AR237:12, AR295:11, AR266:11,
··				AR252:11, AR213:11, AR168:11, AR288:11, AR254:11, AR215:11, AR228:11, AR221:10, AR272:10, AR230:10, AR250:10, AR250:10, AR204:10, AR039:10, AR242:10, AR239:9, AR245:9, AR289:9, AR195:9, AR256:9, AR170:9, AR169:9,
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				L0493:1, L0666:1, S0052:1, H0539:1, L0747:1, L0752:1 and L0366:1.
30	HATDF29	845965	40	AR290:4, AR221:3, AR170:3, AR253:3, AR169:3, AR213:3, AR178:3, AR243:3, AR215:3, AR162:2, AR180:2, AR277:2, AR2
				AR181:1, AR161:1, AR163:1, AR237:1, AR264:1, AR282:1, AR217:1, AR291:1, AR272:1, AR172:1, AR268:1,
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31	HATEE46	565618	41	AR296:15, AR266:6, AR176:6, AR291:6, AR289:6, AR255:5, AR257:5, AR183:5, AR182:5, AR269:5, AR252:4,
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				ARO61:1, AR211:1
				S0126:1, S3014:1, L0740:1, L0747:1, L0750:1, L0756:1, L0752:1, L0759:1, L0599:1 and S0026:1.
32	HAUAI83	639009	42	H0294:2
	HAUAI83	383592	420	

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				H0023:1, H0071:1, H0083:1, T0004:1, T0042:1, L0520:1, L0761:1, L0772:1, L0771:1, L0773:1, L0648:1, L0662:1,
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34	HBAFV19	843036	4	AR196:41, AR173:39, AR164:35, AR166:32, AR165:31, AR262:30, AR162:25, AR161:25, AR163:24, AR174:24,
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	_			AR270:9, AR221:9, AR295:9, AR215:9, AR315:9, AR170:9, AR219:8, AR294:8, AR033:8, AR190:8, AR210:8,
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				AR277:7, AR252:7, AR291:7, AR216:7, AR282:7, AR309:7, AR201:6, AR198:6, AR245:6, AR225:6, AR272:6,
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Gene No:	cDNA Clone ID	Contig ID:	SEQ D NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
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35	HBAMB15	671835	45	AR245:4, AR213:3, AR176:3, AR224:3, AR168:3, AR165:2, AR164:2, AR183:2, AR197:2, AR204:2, AR238:2, AR266:2, AR282:2, AR162:2, AR171:2, AR271:2, AR289:2, AR291:2, AR295:2, AR274:2, AR096:2, AR268:2, AR297:2, AR296:2, AR265:2, AR161:1, AR311:1, AR192:1, AR269:1, AR261:1, AR179:1, AR182:1, AR234:1, AR191:1, AR277:1, AR181:1, AR237:1, AR313:1, AR300:1, AR089:1 H0410:1, H0530:1,
				H0328:1, L0455:1 and L0740:1.
36	HBCPB32	1352403	46	AR277:11, AR283:8, AR219:6, AR104:6, AR282:5, AR316:5, AR240:5, AR055:5, AR039:5, AR089:4, AR185:4, AR313:4, AR300:3, AR096:3, AR218:3, AR299:3, AR060:2. H0663:2, S6028:1 and S0424:1.
	HBCPB32	1045580	421	
37	нвсог32	1134954	47	AR277:25, AR283:20, AR316:17, AR219:16, AR313:15, AR089:14, AR104:14, AR299:14, AR282:13, AR240:13, AR096:13, AR096:
				L0809:3, L0759:3, H0656:2, H0663:2, H0662:2, S0358:2, H0457:2, H0620:2, H0356:2, H0038:2, H0413:2, L0803:2,
				H0658:2, H06/0:2, H0521:2, L0/44:2, L0/54:2, L0/56:2, L0/7/3:2, L0/7/1:2, L0/5/:2, S0436:2, H0423:4, H0263:1, L3673:1, S0114:1, S0442:1, H0730:1, H0747:1, S0476:1, L3089:1, H0600:1, H0586:1, H0574:1, H0486:1, L2570:1,
_				L0021:1, H0253:1, H0251:1, H0024:1, S6028:1, H0179:1, S0214:1, H0428:1, H0030:1, S0440:1, H0641:1, L0637:1,
				L0761:1, L0800:1, L0783:1, L0545:1, L0787:1, L0664:1, L2667:1, H0690:1, L0355:1, H0696:1, H0555:1, L0748:1, L0747:1, L0758:1, S0434:1 and L0597:1.
	HBCQL32	1027748	422	
38	HBGBA69	1352289	48	AR196:22, AR089:21, AR275:21, AR188:20, AR240:19, AR096:19, AR177:18, AR060:18, AR104:18, AR282:18, AR269:17, AR278:17, AR185:17, AR189:16, AR199:15, AR283:15, AR185:15, AR183:15, AR244:15,
				AR218:15, AR219:15, AR186:14, AR299:14, AR248:14, AR247:14, AR211:14, AR197:14, AR173:14, AR254:14,
				AR174:14, AR268:14, AR310:13, AR290:13, AR203:13, AR052:13, AR289:13, AR033:13, AR191:13, AR316:13,
				AR165:13, AR300:13, AR055:13, AR164:12, AR266:12, AR243:12, AR249:12, AR271:12, AR190:12, AR166:12,
				AR273:12, AR270:12, AR241:12, AR178:12, AR253:12, AR061:12, AR173:12, AR252:12, AR246:11, AR181:11, Ap567:11
				AR200:10, AR234:10, AR229:10, AR180:10, AR291:10, AR184:10, AR255:10, AR272:10, AR235:10, AR245:10,
		·		AR192:9, AR161:9, AR296:9, AR039:9, AR221:9, AR231:9, AR163:9, AR251:9, AR201:9, AR257:9, AR236:9,
				AR204:9, AR162:9, AR233:9, AR216:8, AR210:8, AR215:8, AR295:8, AR315:8, AR314:8, AR265:8, AR284:8,
_				AR228:8, AR312:8, AR277:8, AR286:8, AR213:8, AR194:8, AR288:8, AR226:8, AR298:8, AR242:8, AR256:7,
				AR227:7, AR193:7, AR217:7, AR262:7, AR053:7, AR264:7, AR179:7, AR224:7, AR237:6, AR202:6, AR293:6,
				AR230:0, AR214:0, AR29/:0, AR28/:0, AR203:0, AR292:0, AR283:0, AR208:0, AR203:0, AR294:0, AR2233:0,

Cono	CDNA Clone		SEQ	Tissue Distribution Library Code: Count
No.	ID IID	Contig ID:	~	
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				H0653:3, S0358:2, H0733:2, L0717:2, S0278:2, H0318:2, H0309:2, H0357:2, H0150:2, H0687:2, H0181:2, H0413:2,
				H0509:2, L0769:2, L0764:2, L0771:2, L0662:2, L0768:2, L0774:2, L0776:2, L5622:2, L0666:2, L0663:2, L2261:2, ls0126:2, H0718:1, H0713:1, H0740:1, T0049:1, H0657:1, ls0126:2, H0713:1, H0713:1, H0740:1, T0049:1, H0657:1, ls0126:2, H0713:1, H0740:1, H0740:1, H0657:1, ls0126:2, H0740:1, H0740:1, H0657:1, ls0126:2, H0740:1, H0740:1, H0657:1, ls0126:2, H0740:1, H0740:1, H0657:1, ls0126:2, H0667:1, H0740:1, H0740:1, H0667:1, H0667
				S0116:1, S0282:1, H0255:1, H0402:1, H0638:1, S0418:1, S0420:1, S0442:1, S044:1, S0408:1, H0730:1, H0741:1,
				H0735.1, H0776.1, S0300.1, L3388.1, H0370.1, H0592.1, H0643.1, L0623.1, H0156.1, L0021.1, H0253.1, H0263.1,
				L0738:1, H0530:1, H0571:1, H0081:1, H0578:1, H0083:1, H0266:1, H0039:1, H0604:1, H0031:1, H0616:1, H0087:1, H00
	•			10004:1, H0494:1, 50458:1, 50142:1, H0/43:1, H0529:1, L0/50:1, L0/61:1, L0/61:1, L0/63:1, L0/
				H0519:1, H0670:1, H0672:1, H0518:1, S0044:1, H0553:1, H0436:1, S3014:1, L0439:1, L0749:1, L0731:1, L0759:1, S0750:1, H0474:1, S0196:1, H0423:1, and H0506:1.
	HBGBA69	709658	423	
39	HBIAE26	514418	49	AR161:11, AR162:11, AR163:11, AR313:9, AR242:8, AR165:8, AR039:7, AR164:7, AR166:7, AR207:6, AR201:6,
				AR204:6, AR089:6, AR096:6, AR197:6, AR309:6, AR053:5, AR193:5, AR264:5, AR299:5, AR060:5, AR182:5,
				AR173:5, AR185:5, AR198:5, AR236:5, AR300:5, AR181:5, AR228:5, AR271:5, AR176:5, AR277:5, AR055:5,
				AR262:5, AR196:5, AR247:5, AR250:4, AR258:4, AR312:4, AR257:4, AR175:4, AR229:4, AR178:4, AR179:4,
				AR316:4, AR293:4, AR269:4, AR274:4, AR240:4, AR261:4, AR246:4, AR104:4, AR266:4, AR177:4, AR191:4,
				AR233:4, AR275:4, AR192:4, AR268:4, AR183:4, AR213:4, AR205:4, AR231:4, AR297:4, AR288:4, AR174:3,
				AR212:3, AR294:3, AR270:3, AR267:3, AR238:3, AR180:3, AR215:3, AR255:3, AR245:3, AR199:3, AR287:3,
				AR226:3, AR296:3, AR234:3, AR203:3, AR218:3, AR285:3, AR282:3, AR311:3, AR195:3, AR200:3, AR239:3,
				AR283:3, AR263:3, AR217:3, AR2722:3, AR2712:3, AR291:3, AR237:3, AR033:3, AR290:3, AR188:3, AR243:3,
				AK255:5, AK189:5, AK225:5, AK295:5, AK250:5, AK170:5, AK001:2, AK219:2, AK200:2, AK5005:2, AK227:2, Ap325:5, Ap325:3, Ap
				AK230:2, AK232:2, AK210:2, AK130:2, AK171:2, AK203:2, AK211:2, AKZ23:2, AKZ33:1, AKZ33:1, AKZ14:1 50043:1 allu S0146:1.
9	HBIMB51	963208	20	AR225:5, AR162:5, AR161:5, AR223:5, AR224:5, AR170:5, AR180:5, AR176:4, AR183:4, AR214:4, AR163:4,
				AR165:4, AR222:4, AR164:4, AR207:4, AR166:4, AR228:4, AR230:3, AR291:3, AR169:3, AR287:3, AR192:3,
				AR269:3, AR239:3, AR264:3, AR215:3, AR238:3, AR178:3, AR282:3, AR272:3, AR201:3, AR190:3, AR250:3,
				AR173:3, AR297:3, AR234:3, AR267:3, AR257:3, AR197:3, AR168:3, AR288:3, AR181:3, AR263:3, AR245:3,
				AR195:2, AR188:2, AR193:2, AR262:2, AR179:2, AR182:2, AR268:2, AR216:2, AR172:2, AR255:2, AR236:2,
				AR231:2, AR253:2, AR247:2, AR296:2, AR277:2, AR266:2, AR2290:2, AR174:2, AR295:2, AR233:2,
				AR289:2, AR270:2, AR312:2, AR227:2, AR203:2, AR240:2, AR240:2, AR175:2, AR212:2, AR294:2, AR237:2,

Gene No.	cDNA Clone	Contig ID:	SEQ ID	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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	HBIMB51	672711	424	
41	HBINS58	1352386	51	AR222:31, AR214:31, AR169:26, AR223:23, AR235:22, AR224:22, AR283:21, AR195:20, AR170:20, AR168:20, AR264:20, AR263:19, AR212:19, AR207:18, AR282:18, AR161:18, AR311:18, AR172:17, AR089:17,
		•		AR162:16, AR216:16, AR217:16, AR316:16, AR261:16, AR281:16, AR171:16, AR163:16, AR277:16, AR236:14,
				AR104:14, AR309:14, AR213:13, AR308:13, AR096:13, AR314:13, AR240:13, AR055:12, AR310:12, AR299:12, AR104:13, AR265:12, AR265:11, AR205:11, AR202:11, AR202
				AR295;11, AR280;11, AR198;11, AR245;11, AR165;11, AR039;11, AR166;11, AR060;11, AR193;10, AR297:10,
				AR271:10, AR164:10, AR252:10, AR232:10, AR192:10, AR284:10, AR300:10, AR17:10, AR218:10, AR285:10,
				AR221.9, AR254.9, AR262.9, AR181.8, AR204.8, AR291.8, AR275.8, AR185.8, AR243.8, AR274.8, AR286.8,
				AR247.8, AR241.8, AR238.8, AR266.8, AR287.7, AR229.7, AR292.7, AR230.7, AR268.7, AR251.7, AR211.7,
				AR239.7, AR178.7, AR270.7, AR231.7, AR226.7, AR227.7, AR183.7, AR184.7, AR215.6, AR293.6, AR234.6,
_				AR269:6, AR253:6, AR199:6, AR176:6, AR210:6, AR180:6, AR200:6, AR298:6, AR188:6, AR4230:0, AR427:5,
				AR23333, AR2943, AR1733, AR2073, AR2073, AR1913, AR1913, AR1973, AR2963, AR1923, AR2663, AR1923, AR2503, AR2703, AR270
<u></u>	-			ARZ/3:3, ARI/3:3, ARZ/26:3, ARZ/3:3, ARZ/3:3, ARZ/3:1, ARZ/3:3, ARI/3:3, ARI/3:3, ARZ/46:3, ARZ/44:3 H0593:2, H0617:1, L0657:1 and L0592:1.
	HBINS58	961712	425	
	HBINS58	892924	426	
42	HBJID05	1130660	52	AR192:7, AR161:4, AR162:4, AR163:4, AR193:4, AR165:4, AR308:4, AR309:4, AR312:4, AR164:4, AR282:4, AR166:4, AR195:3, AR295:3, AR245:3, AR250:3, AR170:3, AR215:3, AR264:3, AR311:3, AR1176:3, AR201:2,
				AR274.2, AR275.2, AR246.2, AR197.2, AR213.2, AR313.2, AR287.2, AR212.2, AR172.2, AR272.2, AR225.2,
				AR205:2, AR296:2, AR243:2, AR033:2, AR267:2, AR089:2, AR233:2, AR299:2, AR239:2, AR173:2, AR289:2,
:				AR257:2, AR291:2, AR182:2, AR300:1, AR185:1, AR177:1, AR262:1, AR293:1, AR169:1, AR247:1, AR277:1,
	HBJID05	544980	427	AKUOU:1, AKZOS:1, AKUO1:1, AKI 73:1, AKI 71:1, AKZII:1, AKZII:1, AKZII:1
43	HBJJU28	561723	53	AR161:16, AR162:16, AR163:16, AR242:12, AR313:12, AR264:9, AR247:9, AR233:8, AR238:8, AR165:8,
!				AR177:8, AR193:8, AR269:8, AR164:8, AR240:8, AR270:7, AR166:7, AR198:7, AR268:7, AR229:7, AR300:7,

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Gene No:	cDNA Clone	Contig ID:	UO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
				AR309:5, AR271:5, AR207:5, AR173:5, AR263:5, AR267:5, AR189:5, AR192:5, AR291:5, AR287:4, AR185:4, AR295:4, AR175:4, AR190:4, AR286:4, AR285:4, AR288:4,
				AR196:4, AR261:4, AR246:4, AR061:4, AR235:4, AR289:3, AR195:3, AR282:3, AR311:3, AR294:3, AR277:3,
				AR227:3, AR258:3, AR199:3, AR205:3, AR200:3, AR172:3, AR316:3, AR243:3, AR033:3, AR255:3, AR308:3,
				AR232:3, AR203:3, AR188:3, AR225:3, AR039:3, AR295:3, AR297:3, AR250:3, AR060:3, AR216:3, AR168:2, AR104:2, AR283:2, AR252:1, AR171:1, AR217:1, AR254:1, AR214:1, AR260:1, H0318:1 and S0386:1,
44	HBJNC59	1125802	54	AR268:41, AR290:25, AR267:21, AR270:19, AR180:19, AR245:17, AR269:17, AR096:15, AR183:15, AR177:13,
				AR182:12, AR271:12, AR240:11, AR242:11, AR234:11, AR246:11, AR283:11, AR173:10, AR176:10, AR192:10,
				AKZ12:10, AK101:10, AKZ29:3, AK190:3, AK191:3, AKZ13:3, AK109:3, AK173:0, AK200:0, AK173:1, AK170:1, AK170:1, AP100:7 AR228:7 AR300:7 AR303:7 AR303:7 AR301:7 AR300:7 AR300:7 AR3031:7 AR378:6 AR361:6
				AR162:6, AR195:6, AR165:6, AR163:6, AR261:6, AR258:6, AR237:6, AR201:6, AR188:6, AR299:6, AR252:5,
				AR282:5, AR257:5, AR203:5, AR039:5, AR196:5, AR274:5, AR247:5, AR266:5, AR226:5, AR243:5, AR204:4,
				AR255:4, AR170:4, AR165:4, AR230:4, AR164:4, AR200:4, AR166:4, AR207:4, AR295:4, AR288:4, AR300:4,
				AR313:4, AR233:3, AR285:3, AR294:3, AR316:3, AR185:3, AR168:3, AR053:3, AR217:3, AR277:3, AR033:3,
				AR210:3, AR236:3, AR263:2, AR232:2, AR212:2, AR312:2, AR293:2, AR089:2, AR261:2, AR264:2,
				AR311:2, AR222:2, AR171:2, AR227:2, AR205:2, AR214:2, AR211:2, AR216:2, AR060:2, AR291:2, AR287:1,
				[AR172:1, AR308:1, AR104:1, AR223:1, AR219:1 H0521:26, H0522:16, S0360:13, H0255:1, L0775:1, S0374:6, H10445-4, S0476:1, H1045-4, H1059-4, H1047-4, H1057-4, H1057-4, H1057-4, H1057-4, H1057-4, H1058-7-3
				MO44333, 3040633, MO3613, L070633, 3040433, MO03634, MO4273, MO373, MO5173, L07073, L07073, L060634, MO3673, H HAMA33 HA1343 HAA873 SA4383 TA6593 HA6733 TA493 HA5063 SA1163 HA543 HA5433 HAS43
				\$0376.2, H0637.2, L3071.2, \$0280.2, H0706.2, H0120.2, H0318.2, H0327.2, H0455.2, H0424.2, H0100.2, \$0440.2,
				H0649:2, L0769:2, L0774:2, L0776:2, L0657:2, L0547:2, L0783:2, S0292:2, H0555:2, L0754:2, L0747:2, L0750:2,
				L0777:2, S0436:2, L0603:2, H0717:1, H0716:1, H0583:1, H0663:1, S0356:1, S0444:1, L3649:1, H0741:1, L2831:1,
				L3388:1, H0411:1, S6022:1, H0550:1, H0455:1, H0602:1, H0632:1, T0082:1, H0309:1, H0009:1, H0015:1, H0510:1,
				H0375:1, H0687:1, H0039:1, H0030:1, H0031:1, S0294:1, H0509:1, H0641:1, H0647:1, H0538:1, L0762:1, L0763:1,
				[155551] L077211 L064811 L064811 L035511 L035111 L035111 L055111 L062811 L063811 L0638
				L0540:1, L0543:1, H0689:1, S0380:1, S0332:1, S0044:1, S0406:1, L0733:1, S0260:1, S0434:1, H0633:1, L2367:1 and H0352:1.
	HBJNC59	899397	428	
	HBJNC59	902207	429	

			CEO	
Gene No:	ਹ	Contig ID:	NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
45	нвмсі50	668268	55	AR170:3, AR180:3, AR172:3, AR254:2, AR271:2, AR223:2, AR221:2, AR225:2, AR178:2, AR283:2, AR274:2, AR282:2, AR089:2, AR203:1, AR205:1, AR176:1, AR311:1, AR291:1, AR272:1, AR161:1, AR275:1, AR222:1, AR243:1, AR242:1, AR286:1 H0421:1
46	HBNAW17	526797	99	AR266:6, AR245:3, AR168:2, AR246:2, AR217:2, AR177:2, AR291:2, AR264:2, AR274:1, AR165:1, AR267:1, AR312:1, AR216:1, AR311:1, AR164:1, AR261:1, AR182:1, AR299:1, AR257:1, AR166:1, AR243:1, AR309:1, AR089:1, AR224:1, AR175:1 L0766:3 and H0188:1.
47	HBXFL29	842802	57	AR243:4, AR275:3, AR213:3, AR197:2, AR268:2, AR283:2, AR240:2, AR183:2, AR264:2, AR039:2, AR225:2, AR223:2, AR271:2, AR172:2, AR205:1, AR206:1, AR29:1, AR266:1, AR089:1, AR311:1, AR225:2, AR223:2, AR277:1, AR216:1, AR206:1, AR245:1, AR245:1, AR162:1, AR266:1, AR089:1, AR201:1, AR167:1, AR290:1, AR290:1, AR290:1, AR201:1, AR201:1, AR200:1, AR290:1, AR290:1, AR200:1, AR20
48	HCACU58	625923	58	AR170:4, AR225:4, AR197:3, AR253:3, AR183:3, AR242:3, AR270:2, AR311:2, AR266:2, AR275:2, AR168:2, AR172:2, AR223:2, AR282:2, AR291:2, AR169:2, AR272:2, AR198:1, AR096:1, AR240:1, AR269:1, AR283:1, AR192:1, AR164:1, AR300:1, AR224:1, AR252:1 H0341:1, H0125:1, H0580:1, L0747:1 and L0749:1.
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55	HCEFB80	1143407	83	H0052:6, L0439:5, L0794:3, L0748:3, L0415:2, H0661:2, H0559:2, S0049:2, H0327:2, S0051:2, H0399:2, S0036:2,
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99	HCEGR33	425212	99	AR221:5, AR225:4, AR313:4, AR180:4, AR162:4, AR161:4, AR165:4, AR163:4, AR271:3, AR164:3, AR166:3,
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57	HCEWE17	941941	<i>L</i> 9	AR172:5, AR198:5, AR181:5, AR171:5, AR266:4, AR216:4, AR205:4, AR214:4, AR225:4, AR264:4, AR162:4,
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Tissue Distribution Library Code:Count (see Table 4 for Library Codes)			AR253:8, AR053:6, AR196:6, AR198:5, AR191:5, AR313:5, AR245:4, AR181:4, AR174:4, AR195:4, AR189:3, AR096:3, AR089:3, AR214:3, AR214:3, AR247:3, AR247:3, AR248:3, AR2	AR188:2, AR275:2, AR175:2, AR226:2, AR165:2, AR171:2, AR312:2, AR179:2, AR162:2, AR180:2, AR164:2,	AR299:2, AR161:2, AR163:2, AR257:2, AR238:2, AR166:2, AR240:2, AR185:2, AR268:2, AR207:2, AR223:2,	AR199:2, AR060:2, AR178:2, AR316:2, AR204:2, AR173:2, AR295:2, AR200:2, AR183:2, AR212:2, AR309:2, Ap33:3, Ap34:1	AR233:2, AR210:2, AR229:1, AR239:1, AR237:1, AR230:1, AR230:1, AR238:1, AR182:1, AR333:1, AR387:1, AR287:1,	AR283:1, AR282:1, AR266:1, AR232:1, AR252:1, AR230:1 H0052:2, H0261:1, H0271:1 and S0458:1.	AR214:5, AR216:4, AR215:4, AR269:4, AR217:3, AR232:3, AR193:3, AR297:3, AR286:3, AR245:3, AR176:3,	AR294:3, AR264:3, AR197:3, AR295:3, AR200:2, AR312:2, AR096:2, AR165:2, AR104:2, AR263:2, AR183:2, AB154:3, AB33:3, AB306:1	AR104:2, AR243:2, AR108:2, AR127:2, AR234:1, AR261:1, AR289:1, AR309:1, AR237:1, AR308:1, AR293:1, AR293:1	20748;6, L0750:4, S0386:3, L0439:3, L0777:3, H0624:2, H0052:2, L0435:2, L0598:2, L0809:2, L0751:2, L0747:2,	.0756:2, L0753:2, L0731:2, H0422:2, L0718:2, H0265:1, H0381:1, H0459:1, S0356:1, S0360:1, H0619:1, H0393:1,	H0411:1, H0050:1, L0455:1, H0412:1, S0344:1, L0769:1, L0638:1, L0764:1, L0771:1, L0803:1, L0804:1, L0805:1,	.0776:1, L0438:1, H0689:1, H0659:1, H0658:1, H0660:1, H0666:1, L0594:1 and S0100:1.	AR223:4, AR225:3, AR039:3, AR213:3, AR252:2, AR282:2, AR171:2, AR308:2, AR166:2, AR195:2, AR207:1, AP277:1, AP275:1, AP206:1, AP207:1,	AR199:1, AR162:1 H0196:1	AR282:5, AR060:5, AR309:4, AR055:4, AR266:4, AR162:4, AR213:4, AR161:4, AR163:4, AR225:4, AR254:3,	AR270:3, AR177:3, AR207:3, AR300:3, AR176:3, AR089:3, AR192:3, AR263:2, AR221:2, AR172:2, AR198:2,	AKIU4:2, AK224:2, AK203:2, AK240:2, AK211:2, AK103:2, AK103:2, AK210:2, AK104:2, AK171:2, AK171:2. AK217:2. A R R R S S S S S S S S S S S S S S S S	AR183-2. AR308-2. AR257-2. AR039-2. AR296-2. AR272-2. AR264-1. AR033-1. AR261-1. AR311-1. AR246-1.	AR212:1, AR286:1, AR289:1, AR255:1, AR231:1, AR237:1, AR061:1, AR179:1, AR238:1, AR297:1, AR245:1,	AR195:1, AR215:1 L0794:3, L0764:2, L0439:2, H0052:1, H0597:1, T0006:1, L0766:1, H0648:1, S0330:1 and			
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Contig ID:	893535	460407	543370						636078							740781		1016919						863677	874128	
Gene cDNA Clone Contig ID: NO: No:	HCEWE17	HCEWE17	HCEWE20						HCGMD59		-	-				HCMSQ56		HCNDR47						HCNDR47	HCNDR47	
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64	HCUIM65	550208	74	AR223:4, AR215:3, AR268:3, AR270:3, AR260:3, AR161:3, AR246:3, AR162:3, AR166:2, AR171:2, AR254:2, AR217:2, AR213:2, AR213:2, AR230:2, AR250:2, AR257:2, AR269:2, AR268:1, AR313:1, AR179:1,
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				S0152:1, H0696:1, S0404:1, S0037:1, L0746:1, L0779:1, S0031:1, H0707:1, S0434:1, L0480:1, L0608:1, L0604:1,
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99	HCWEB58	1352416	9/	AR171:4, AR309:3, AR252:3, AR215:3, AR266:3, AR235:2, AR310:2, AR251:2, AR221:2, AR169:2, AR224:2,
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Gene	cDNA Clone	Contig ID:	SEQ ID	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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	HCWEB58	889268	440	
29	HCWKC15	553621	LL	AR313:9, AR164:8, AR165:8, AR166:8, AR163:7, AR161:7, AR162:7, AR089:6, AR039:5, AR173:5, AR096:5, AR180:5, AR192:4, AR263:4, AR299:4, AR282:4, AR242:4, AR053:4, AR178:4, AR175:4, AR247:4, AR269:4, AR269:4, AR269:4, AR269:4, AR269:3, AR2
				AR290.3, AR243.3, AR264.3, AR185.3, AR300.3, AR179.3, AR311.3, AR191.3, AR293.3, AR181.3, AR272.3,
				AR297.3, AR213.3, AR171.3, AR270.3, AR183.3, AR238.3, AR236.3, AR316.3, AR060.3, AR308.3, AR294.3,
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				AR218:2, AR267:2, AR182:2, AR228:2, AR268:2, AR204:2, AR190:2, AR246:2, AR239:2, AR232:2, AR261:2,
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				AR227:1, AR230:1 H0305:2 and H0589:1.
89	HCWLD74	628256	78	AR268:4, AR243:3, AR270:3, AR180:3, AR171:3, AR282:3, AR162:3, AR254:3, AR252:2, AR039:2, AR204:2,
3				AR238:2, AR161:2, AR170:2, AR269:2, AR267:2, AR257:2, AR210:2, AR168:2, AR262:2, AR053:2, AR183:2,
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				AR201:1 H0305:3 and H0589:1.
9	HCYBG92	598019	79	AR215:7, AR168:7, AR170:7, AR172:6, AR248:6, AR254:5, AR235:5, AR225:5, AR245:5, AR171:5, AR169:5,
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Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
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70	HDHEB60	499233		AR195:10, AR245:9, AR242:9, AR309:9, AR196:8, AR225:8, AR198:8, AR207:8, AR246:8, AR169:8, AR170:8, AR223:8, AR224:7, AR214:7, AR039:7, AR172:7, AR215:7, AR201:7, AR222:7, AR193:7, AR205:7, AR201:7, AR199:7, AR204:7, AR208:7, AR172:7, AR201:7, AR201:7, AR202:7, AR164:6, AR205:7, AR201:7, AR199:7, AR204:6, AR201:7, AR201:7, AR201:7, AR201:7, AR201:7, AR201:7, AR201:7, AR201:7, AR201:5, AR201:6, AR201:6, AR201:5, AR201:4, AR201:2, AR201:3, AR201:3, AR201:3, AR201:3, AR201:3, AR201:3, AR201:2, AR201:2, AR201:1, AR
7.7	HDHMA45	902513	81	AR225:9, AR217:8, AR2124:8, AR225:8, AR215:8, AR105:1, AR106:9, AR110:0, AR110:0, AR110:0, AR110:0, AR110:0, AR225:9, AR222:5, AR222:6, AR222:4, AR288:5, AR310:5, AR311:5, AR207:4, AR222:4, AR288:5, AR310:3, AR311:5, AR207:3, AR209:3, AR209:2, AR209:3, AR209:2, AR200:2, AR
	HDHMA45	812764	441	

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Gene No:	cDNA Clone ID	Contig ID: NO:	UO:X	
72	428 428	1062783	83	AR254:26, AR264:22, AR248:18, AR251:17, AR240:17, AR313:16, AR268:14, AR272:13, AR265:27, AR184:26, AR254:26, AR264:22, AR248:18, AR251:17, AR240:17, AR313:16, AR268:14, AR272:13, AR290:13, AR311:13, AR310:13, AR310:13, AR312:9, AR308:19, AR368:14, AR257:10, AR308:10, AR275:10, AR308:19, AR247:9, AR312:9, AR316:7, AR198:7, AR274:9, AR312:9, AR308:9, AR368:9, AR271:8, AR177:8, AR270:8, AR267:8, AR316:7, AR198:7, AR252:7, AR244:7, AR244:7, AR242:6, AR163:6, AR163:6, AR165:6, AR162:6, AR162:6, AR161:6, AR242:6, AR163:6, AR300:5, AR197:5, AR299:6, AR162:6, AR166:6, AR201:6, AR2625:7, AR244:7, AR243:7, AR185:4, AR061:4, AR089:4, AR299:6, AR197:5, AR282:5, AR282:5, AR282:3, AR282:3, AR202:3, AR203:3, AR282:3, AR283:3, AR282:3, AR282:2, AR292:2, AR292:1, A
	HDPBA28	866429	442	
73	HDPCL63	1019008	83	AR295:12, AR202:15, AR194:15, AR196:14, AR315:13, AR207:13, AR206:13, AR265:13, AR205:12, AR244:12, AR195:12, AR222:11, AR033:11, AR235:10, AR214:10, AR263:10, AR218:10, AR246:10, AR197:10, AR261:10, AR284:10, AR311:19, AR197:10, AR261:10, AR284:10, AR311:19, AR197:10, AR261:10, AR284:10, AR310:10, AR217:10, AR242:10, AR291:19, AR193:19, AR192:9, AR291:9, AR192:9, AR292:9, AR291:9, AR193:8, AR292:9, AR292:9, AR292:9, AR292:9, AR292:9, AR292:9, AR193:8, AR292:9, AR292:17, AR292:7, AR292:9, AR292:5, AR292:3, AR29

Gene	cDNA Clone		SEQ ID	Tissue Distribution Library Code: Count
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				L0800:1, L0764:1, L0648:1, L0768:1, L0774:1, L0776:1, L0657:1, L0559:1, L0519:1, L0789:1, L0792:1, L0666:1, L0664:1, L0709:1, L0644:1, L0709:1, L07
				H0436:1, L0748:1, L0780:1, L0759:1, L0601:1, L0366:1 and H0423:1.
	HDPCL63	847045	443	
	HDPCL63	897484	444	
74	HDPC025	460682	84	AR060:2, AR055:2, AR282:2 H0521:2, H0445:2, H0394:1, H0747:1, H0581:1, L0761:1 and L0750:1.
75	HDPCY37	837699	82	AR215:26, AR214:25, AR263:23, AR197:22, AR207:22, AR217:19, AR195:19, AR212:19, AR169:19, AR222:18,
				AR168:18, AR269:17, AR243:17, AR216:16, AR172:16, AR264:16, AR225:16, AR171:16, AR224:16, AR223:16,
				AR311:16, AR221:15, AR253:15, AR165:15, AR198:15, AR246:15, AR164:15, AR192:15, AR277:14, AR240:14,
				AR170:14, AR166:14, AR162:14, AR161:14, AR213:14, AR163:13, AR096:13, AR245:13, AR089:13, AR299:13,
				AR242:13, AR309:12, AR183:12, AR316:12, AR308:12, AR219:12, AR193:12, AR312:12, AR201:12, AR235:12,
				AR313:12, AR250:12, AR196:11, AR282:11, AR205:11, AR283:11, AR053:11, AR236:11, AR275:11, AR291:10,
				JAR295:10, AR272:10, AR252:10, AR218:10, AR270:10, AR185:10, AR288:10, AR268:10, AR261:10, AR039:10,
				AR297:9, AR199:9, AR247:9, AR285:9, AR173:9, AR033:9, AR060:9, AR191:9, AR177:9, AR174:9, AR175:9,
				AR290:9, AR181:8, AR300:8, AR238:8, AR211:8, AR210:8, AR286:8, AR176:8, AR188:8, AR182:8, AR287:8,
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				AR227:4, AR237:4, AR260:4, AR230:4 S0440:6, L0766:4, L3659:3, H0052:3, L0662:3, L0776:3, L0666:3, L0665:3,
				H0521:3, S0476:2, H0438:2, H0581:2, H0263:2, H0494:2, L0763:2, L0770:2, L0769:2, L0649:2, L0659:2, L0664:2,
				L2261:2, L3829:2, L0748:2, L0439:2, L0747:2, S0436:2, H0265:1, H0556:1, S0440:1, H0717:1, S0444:1, S0278:1,
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				H0553:1, H0412:1, H0646:1, S0002:1, L0796:1, L3905:1, L0644:1, L0764:1, L0774:1, L0376:1, L0806:1, L0654:1,
				L0807:1, L0383:1, L3841:1, L2651:1, L2263:1, L2260:1, L2262:1, S0126:1, H0684:1, H0435:1, H0478:1, S0028:1,
	HDPCY37	604114	445	LOVO1:1, LOVO4:1, LOVO4:1, LOVO:1, LOVO:1, LOVO3:1, MOCHO:1, MOCHO:1, MOCHO:1
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9/	HDPFB02	898208	98	AR194:75, AR202:65, AR244:55, AR206:49, AR241:35, AR265:28, AR273:25, AR310:25, AR281:22, AR243:21,
				AR205:21, AR246:20, AR292:19, AR052:19, AR315:18, AR204:18, AR198:17, AR251:16, AR192:15, AR280:15,
				AR314:14, AR2/1:14, AR284:13, AR193:13, AR222:13, AR186:13, AR263:15, AR039:13, AR282:12, AR248:12,

Tissue Distribution Library Code: Count (see Table 4 for Library Codes)	AR171:12, AR169:12, AR232:12, AR213:12, AR033:11, AR247:11, AR299:11, AR242:10, AR312:10, AR033:10, AR309:10, AR275:10, AR276:10, AR275:10, AR276:10, AR276:			AR194:31, AR202:28, AR198:25, AR205:24, AR206:24, AR281:24, AR246:22, AR244:21, AR263:21, AR315:20, AR241:19, AR192:19, AR243:19, AR282:18, AR033:17, AR280:17, AR265:17, AR275:16, AR283:16, AR273:15, AR204:15, AR285:14, AR291:14, AR277:14, AR296:14, AR247:14, AR314:13, AR284:13, AR284:13, AR286:13, AR286:13, AR286:13, AR286:13, AR286:11, AR316:11, AR286:12, AR104:12, AR316:12, AR316:11, AR300:11, AR300:11, AR331:10, AR286:11, AR286:11, AR300:11, AR286:11, AR286:10, AR218:10, AR286:10, AR298:10, AR298:
SEQ ID NO:X		446	447	87
Contig ID:		1056541	997408	588697
cDNA Clone Contig ID:		HDPFB02	HDPFB02	НЪРҒҒ39
Gene No:				77

Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
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78	HDPFP29	628254	88	AR311:15, AR263:15, AR223:14, AR224:14, AR214:14, AR195:13, AR215:12, AR222:12, AR168:12, AR309:12, AR263:15, AR263:11, AR263:11, AR263:11, AR275:11, AR275:10, AR215:10, AR215:10, AR275:11, AR275:11, AR275:10, AR215:10, AR215:10, AR275:10, AR215:10, AR275:10, AR275:
6/	HDPGT01	771583	68	AR268:5, AR244:4, AR282:3, AR251:3, AR242:3, AR241:3, AR052:3, AR184:2, AR271:2, AR310:2, AR176:2, AR194:2, AR039:2, AR309:2, AR283:1, AR178:1, AR289:1, AR217:1, AR277:1, AR277:1, AR284:1, AR221:1, AR226:1, AR266:1, H0521:3, S0278:2, S0222:2, H0284:2, H0265:1, H0728:1, S0007:1, H0208:1, H0586:1, H0497:1, H0642:1, H0581:1, H0652:1, H0672:1, H0672:1, H0673:1, H06
08	HDPHI51	460679	06	AR195:9, AR192:9, AR207:9, AR215:8, AR264:8, AR225:7, AR263:7, AR311:7, AR168:7, AR309:7, AR252:6, AR172:6, AR245:6, AR246:6, AR246:6, AR246:6, AR245:6, AR275:6, AR245:6, AR246:6, AR246:5, AR246:4, AR246:4, AR246:4, AR346:4, AR346:4, AR346:4, AR346:4, AR346:4, AR346:4, AR346:4, AR246:4, AR2

Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
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81	HDPJF37	704487	91	AR215:15, AR225:14, AR253:11, AR213:10, AR221:9, AR221:3, AR217:8, AR196:8, AR311:8, AR212:8, AR214:8, AR218:7, AR256:7, AR053:7, AR309:7, AR216:7, AR291:7, AR096:7, AR219:7, AR254:7, AR165:7, AR256:7, AR164:7, AR164:7, AR162:7, AR269:6, AR172:6, AR163:6, AR204:6, AR204:7, AR204:6, AR204:7, AR204:4, AR204:4, AR204:4, AR204:4, AR204:4, AR204:4, AR206:4, AR206:4, AR206:4, AR204:4, AR204:1,
83	нDРЈМ30	879325	92	AR268:8, AR289:6, AR184:6, AR266:5, AR223:5, AR169:5, AR290:4, AR286:4, AR224:4, AR194:4, AR268:8, AR289:6, AR184:6, AR266:5, AR223:5, AR169:5, AR290:4, AR286:4, AR2824:4, AR194:4, AR275:4, AR210:4, AR216:3, AR282:3, AR286:3, AR286:2, AR286:2, AR286:2, AR286:2, AR286:2, AR286:2, AR286:2, AR286:2, AR286:2, AR286:3, AR286:3, AR286:3, AR286:3, AR286:3, AR286:3, AR286:3, AR286:3, AR286:1, AR198:1, AR
	HDPJM30	603517	448	
83	HDPMM88	972734	1	AR202:35, AR096:34, AR194:33, AR206:31, AR24:25, AR241:22, AR268:21, AR281:20, AR290:19, AR265:17, AR315:15, AR184:15, AR246:15, AR310:14, AR192:13, AR269:12, AR270:12, AR282:12, AR243:11, AR314:11,

	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)	AR280:11, AR267:10, AR292:10, AR183:9, AR263:9, AR299:9, AR284:9, AR198:9, AR055:8, AR205:8, AR251:8, AR271:8, AR271:8, AR206:8, AR313:8, AR298:8, AR033:8, AR033:8, AR204:7, AR052:7, AR277:7, AR177:7, AR238:7, AR234:7, AR061:6, AR247:6, AR295:6, AR104:6, AR300:6, AR285:6, AR089:6, AR316:6, AR186:6, AR185:6, AR240:5, AR053:5, AR249:5, AR249:5, AR271:5, AR271:5, AR291:5, AR289:5, AR182:5, AR112:5, AR175:4,	AR253:4, AR229:4, AR248:4, AR232:4, AR309:4, AR215:4, AR226:4, AR274:4, AR219:4, AR286:4, AR296:4, AR227:4, AR237:4, AR218:4, AR259:3, AR295:3, AR294:3, AR294:3, AR242:3, AR179:3, AR293:3, AR060:3, AR27:4, AR27:4, AR28:4,	AR170:3, AR193:3, AR233:3, AR169:2, AR224:2, AR256:2, AR257:2, AR258:2, AR171:2, AR217:2, AR172:2, AR170:3, AR195:1, AR308:1, AR308:1, AR263:1, AR162:1, AR162:1, AR169:1, AR208:1, L0754:2, L0777:2, H0717:1, H0740:1, S0212:1, S0360:1, S0408:1, H0747:1, H004:1, H0581:1, L0142:1, H0674:1, H0646:1, S0422:1, L0809:1, L0787:1, H0521:1 and H0522:1.							AR263:16, AR295:6, AR292:6, AR248:6, AR313:6, AR033:6, AR246:6, AR251:6, AR282:5, AR283:5, AR296:7, AR295:6, AR292:6, AR248:6, AR313:6, AR033:6, AR246:6, AR251:6, AR282:5, AR283:5, AR096:5, AR247:5, AR244:5, AR249:5, AR312:5, AR241:4, AR295:4, AR218:4, AR213:4, AR213:4, AR234:4, AR294:4, AR277:4, AR259:4, AR300:4, AR219:4, AR205:3, AR209:4, AR213:4, AR232:4, AR215:3, AR039:3, AR277:3, AR053:3, AR283:3, AR293:3, AR293:3, AR271:3, AR284:3, AR284:3, AR283:3, AR284:3, AR286:2, AR243:3, AR286:2, AR266:2, AR266:2, AR286:2, AR286:3, AR286:3, AR286:3, AR386:2, AR286:1, AR291:1, AR291:1, AR296:1, AR291:1, AR296:1, AR291:1, AR296:1, AR291:2, AR296:3, H0543:3, H0543:3, H0422:3, H0341:2, L0763:2, L0770:2, L0763:2, L0770:2, L0763:2, L0770:2, L0763:2, L0763:1, H0323:1, H0492:1, S0040:1, H0022:1, H0327:1, H0323:1, H0492:1, S0040:1, H0032:1, T0110:1, H0320:1, H0545:1, L0494:1, H0560:1, H0033:1, H0043:1, L0763:1, H0492:1, S0040:1, H0032:1, H0331:1, H0492:1, S0040:1, H0032:1, H0320:1, H0250:1, H0250:1, H0055:1, L0065:1, H0053:1, L0065:1, H0053:1, H0050:1, H0033:1, L0060:1, H0032:1, H0050:1, H0052:1, H0050:1, H0
SEQ	ID NO:X				449	450	451	452	453	454	46
	Contig ID: NO:				906121	902299	885059	874074	854246	854245	897276
	Gene cDNA Clone				HDPMM88	HDPMM88	HDPMM88	HDPMM88	HDPMM88	HDPMM88	нрроез2
	Gene No:										8

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Gene No:	cDNA Clone ID	Contig ID:	NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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82	HDPOH06	683371	56	AR272:69, AR212:53, AR214:43, AR311:39, AR274:36, AR245:35, AR165:33, AR216:32, AR308:32, AR166:31, AR161:30, AR1212:53, AR214:43, AR311:39, AR272:69, AR165:28, AR165:27, AR308:27, AR308:27, AR171:26, AR223:25, AR053:25, AR252:23, AR264:29, AR168:23, AR174:22, AR225:21, AR169:21, AR309:27, AR171:26, AR223:25, AR053:25, AR252:23, AR264:29, AR168:23, AR745:11, AR245:11, AR205:21, AR308:25, AR197:20, AR172:19, AR263:18, AR275:18, AR247:17, AR254:17, AR221:17, AR170:17, AR313:15, AR185:15, AR189:15, AR199:15, AR236:11, AR236:11, AR261:14, AR256:13, AR246:13, AR239:10, AR262:9, AR177:9, AR200:9, AR300:9, AR255:9, AR199:19, AR295:8, AR295:9, AR191:7, AR181:7, AR295:7, AR191:7, AR181:7, AR295:7, AR295:6, AR295:6, AR295:6, AR295:5, AR196:4, AR295:5, AR295:3, AR296:3, AR296:3, AR296:3, AR295:3, AR295:3, AR295:3, AR295:3, AR295:3, AR296:3, AR296:3
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98	HDPOJ08	731863	96	AR250:19, AR254:19, AR269:19, AR162:9, AR164:9, AR163:9, AR181:3, AR270:12, AR255:12, AR183:10, AR267:10, AR180:10, AR161:9, AR162:9, AR165:9, AR164:9, AR163:9, AR181:3, AR166:8, AR173:8, AR174:8, AR184:7, AR235:7, AR252:7, AR229:7, AR272:7, AR176:7, AR177:6, AR181:8, AR265:6, AR239:6, AR182:6, AR1828:5, AR283:7, AR236:4, AR239:5, AR288:5, AR287:5, AR287:5, AR287:5, AR285:4, AR280:5, AR280:5, AR179:5, AR228:5, AR236:4, AR286:4, AR286:3, AR286:2, AR286:2, AR286:2, AR286:2, AR286:2, AR286:2, AR286:2, AR286:3, AR286:2, AR286:3, AR286:3, AR286:3, AR286:3, AR286:1, AR286:1, AR286:1, AR286:1, AR286:3, L0588:3, L0770:3, L0766:11, H0521:10, L0803:7, L0748:6, L0717:5, L0759:5, S0003:4, L3832:4, H0663:3, H0156:3, L0598:3, L0770:3,

Tissue Distribution Library Code: Count (see Table 4 for Library Codes)		AR214:47, AR207:47, AR263:40, AR222:34, AR169:33, AR212:31, AR212:31, AR213:30, AR223:29, AR170:29, AR311:29, AR309:28, AR168:28, AR195:27, AR264:26, AR192:26, AR216:24, AR216:24, AR295:24, AR171:24, AR245:24, AR217:23, AR198:22, AR308:22, AR271:22, AR161:21, AR162:21, AR163:21, AR263:20, AR172:23, AR198:22, AR308:22, AR271:22, AR201:20, AR231:19, AR205:19, AR172:23, AR166:20, AR197:20, AR242:20, AR201:20, AR193:19, AR205:11, AR265:11, AR246:17, AR261:21, AR295:17, AR295:17, AR295:17, AR295:17, AR295:17, AR295:17, AR295:11, AR295:11, AR295:11, AR295:11, AR295:13, AR295:13, AR295:13, AR295:13, AR295:13, AR295:13, AR295:11, AR295:1, AR			AR169:8, AR235:5, AR265:5, AR180:4, AR176:4, AR161:4, AR163:4, AR311:4, AR162:4, AR269:3, AR165:3, AR172:3, AR172:3, AR272:3, AR2	
SEQ ID NO:X		66	459	460	0	461
Contig ID:	732097	1309174	1040056	882768	1352280	689129
cDNA Clone Contig ID: NO:X	HDPSB18	HDPSH53	HDPSH53	HDPSH53	HDPSP01	HDPSP01
Gene No:		68			06	

Gene No:	cDNA Clone	SEC Contig ID: NO:3	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
91	HDPSP54	744440	101	AR221231, AR16836, AR214:51, AR169:41, AR224:40, AR222:38, AR223:37, AR195:36, AR235:32, AR217:31, AR168:30, AR172:30, AR311:29, AR63:28, AR192:28, AR196:28, AR171:27, AR198:27, AR213:27, AR2212:31, AR161:26, AR264:26, AR252:26, AR162:25, AR106:25, AR245:24, AR033:23, AR225:23, AR2216:23, AR163:22, AR69:22, AR261:22, AR215:21, AR104:21, AR104:21, AR295:20, AR218:20, AR236:19, AR193:19, AR191:19, AR211:19, AR197:18, AR177:21, AR181:21, AR104:21, AR295:20, AR240:18, AR240:18, AR165:17, AR166:17, AR166:17, AR295:17, AR166:17, AR295:17, AR166:17, AR295:17, AR185:14, AR285:14, AR285:14, AR295:16, AR240:18, AR285:14, AR285:14, AR295:14, AR285:14, AR285:14, AR285:14, AR295:13, AR295:3, AR295:1, L0773:1, L0794:1, L0804:1, L0787:1, L0655:1, L0755:1 and L0758:1, L0773:1, L0794:1, L0804:1, L0787:1, L0755:1, L0758:1, L0773:1, L0794:1, L0804:1, L0787:1, L0755:1, L0758:1, L0773:1, L0794:1, L0804:1, L0787:1, L0758:1, L0758:1, L0773:1, L0794:1, L0804:1, L0787:1, L0758:1, L0773:1, L0794:1, L0794:1, L0787:1, L0794:1, L0794:1, L0795:1, L0795:1, L0794:1, L0794:1, L0795:1, L
	HDPSP54	502472	462	
	HDPUW68	812737	102	AR253:15, AR052:14, AR213:11, AR184:11, AR230:11, AR228:9, AR170:9, AR250:8, AR168:8, AR254:8, AR225:6, AR297:6, AR053:6, AR211:5, AR267:5, AR268:5, AR268:5, AR261:5, AR096:5, AR214:5, AR238:5, AR178:5, AR249:5, AR216:5, AR173:5, AR239:5, AR266:5, AR182:4, AR161:4, AR162:4, AR217:4, AR269:4, AR282:4, AR282:4, AR267:4, AR267:3, AR191:3, AR293:3, AR308:4, AR191:3, AR293:3, AR293:3, AR191:3, AR293:3, AR293:3, AR293:3, AR267:3, AR267:3, AR267:3, AR267:3, AR293:3, AR293:3, AR191:3, AR293:3, AR293:3, AR192:2, AR292:2, AR292:2, AR262:2, AR262:2, AR262:2, AR262:2, AR262:2, AR262:2, AR262:2, AR262:2, AR262:2, AR262:1, AR262:1

Gene 93 93 94	CDNA Clone Contig ID: NO:X HDPVW11 1036997 103 HDPVW11 896530 463 HDPWN93 992925 104	Contig ID: 1036997 896530 992925		Tissue Distribution Library Code: Count (see Table 4 for Library Codes) AR169:4, AR215:4, AR192:3, AR225:3, AR183:2, AR216:2, AR180:2, AR271:2, AR214:2, AR264:2, AR188:3, AR293:1, BO38:1, H057:2, L0777:2, L0777:2, L0777:2, H0713:1, H0484:1, H0253:1, S0408:1, H0649:1, H0699:1, L0640:1, L0763:1, L0770:1, L07
95	HDPWN93 HDPWN93 HDPWN93	887914 905983 879048	464 465 105	AR213:2, AR232:2, AR200:2, AR224:2, AR212:2, AR293:2, AR191:2, AR262:2, AR053:2, AR229:2, AR189:2, AR275:2, AR181:2, AR203:2, AR237:2, AR205:2, AR205:2, AR268:2, AR287:2, AR214:2, AR255:2, AR171:2, AR290:2, AR275:2, AR286:2, AR286:2, AR296:2, AR271:2, AR296:2, AR290:2, AR277:2, AR286:2, AR286:1, AR294:1, AR174:1, AR294:1, AR175:1, AR294:1, AR294:1, AR199:1, AR294:1, AR199:1, AR293:1, AR295:1, AR295:2, AR295:3, AR295:3, AR295:5, AR2

Tissue Distribution Library Code:Count (see Table 4 for Library Codes)	AR282:5, AR168:5, AR313:5, AR222:5, AR240:5, AR204:5, AR261:5, AR193:4, AR312:4, AR104:4, AR224:4, AR246:4, AR176:4, AR055:4, AR299:4, AR171:4, AR283:4, AR277:4, AR277:4, AR174:4, AR316:4, AR178:4, AR295:4, AR053:4, AR205:4, AR205:4, AR237:4, AR237:4, AR277:4, AR300:4, AR257:3, AR178:4, AR295:4, AR293:3, AR293:3, AR283:3, AR285:3, AR297:3, AR295:3, AR293:3, AR293:3, AR293:3, AR285:3, AR295:3, AR295:3, AR296:3, AR296:3, AR297:3, AR296:3, AR296:2, AR199:2, AR199:2, AR199:2, AR199:2, AR199:2, AR199:2, AR199:2, AR298:3, AR298:1, AR2				AR206:6, AR263:4, AR244:3, AR273:3, AR310:2, AR215:2, AR250:2, AR169:2, AR243:2, AR171:2, AR282:2, AR216:2, AR253:2, AR283:2, AR245:2, AR245:2, AR245:2, AR247:2, AR183:2, AR277:2, AR060:2, AR212:1, AR217:1, AR238:1, AR312:1, AR186:1, AR271:1, AR266:1, AR055:1, AR255:1, AR262:1, AR311:1, AR289:1, AR231:1, AR296:1, AR257:1, AR290:1, AR096:1, AR089:1, AR227:1, L0766:5, L0779:2, T0082:1 and L0807:1.		AR242:4, AR246:4, AR250:3, AR263:3, AR195:3, AR272:3, AR264:3, AR170:3, AR282:3, AR215:3, AR163:3, AR162:4, AR246:4, AR246:4, AR250:3, AR198:3, AR165:3, AR161:3, AR197:2, AR266:2, AR053:2, AR169:2, AR212:2, AR205:2, AR243:2, AR243:2, AR240:2, AR240:2, AR270:2, AR221:2, AR296:2, AR213:2, AR216:2, AR214:2, AR29:2, AR247:2, AR060:2, AR164:2, AR267:1, AR297:1, AR183:1, AR217:1, AR172:1, AR286:1, AR179:1, AR179:1, AR297:1, AR311:1, AR311:1, AR313:1, AR288:1, AR271:1, AR188:1, AR268:1, AR269:1, AR308:1, AR287:1, AR287:1, AR311:1, AR313:1, AR287:1, AR287:1, AR308:1, AR287:1, AR287:1, AR397:1, AR308:1, AR287:1, AR287:1, AR397:1, AR308:1, AR288:1, AR173:1, AR368:1, AR287:1, AR398:1, AR308:1, AR308:1, AR308:1, AR308:1, AR287:1, AR297:1, AR033:1, L0439:17, L074:16, L076:2, L0663:7, L0763:3, H0553:3, H0553:3, H0599:3, H0563:2, H0623:3, H0622:3, H0638:2, H0638:2, H0638:2, H0638:2, H0638:2, H0638:2, H0638:2, H0638:2, H0638:2, L076:2, L0649:2, L0774:2, L0677:2, L0666:2, H0144:2, L0566:2, H0774:2, L0658:2, H0713:1, H0774:2, L0666:2, H0144:2, L0566:2, H070:1, H07
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SEC Contig ID: NO:		904768	895716	895715	1309175	834692	972757
cDNA Clone		HDPXY01	HDPXY01	HDPXY01	нронроз	нронроз	HDTBD53
Gene No:					96		97

200	oDNA Clone		SEQ	Tissue Distribution Library Code: Count
No:	TD Contig ID:	Contig ID:	NO:X	
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				L0788:1, L0789:1, S0126:1, H0689:1, H0682:1, H0658:1, H0648:1, S0328:1, H0539:1, H0696:1, S0406:1, L0740:1, L0757:1, L0603:1, H0665:1, S0196:1, H0423:1 and S0460:1.
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86	HDTBP04	1307742	108	AR282:115, AR316:1 S0392:5, H0478:5, H0479:3, H0485:2 and H0486:1.
	HDTBP04	543618	471	
66	HDTBV77	628582	109	AR183:7, AR184:5, AR269:4, AR207:4, AR245:4, AR270:4, AR182:4, AR214:4, AR172:4, AR223:4, AR263:3,
				AR272:3, AR180:3, AR176:3, AR268:3, AR309:3, AR175:3, AR164:3, AR282:3, AR166:3, AR222:3, AR222:3, AR222:3, AR309:3, AR3
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				AR286.2, AR181.2, AR212.2, AR287.2, AR173.2, AR221.2, AR039.2, AR163.2, AR200.2, AR061.2, AR170.2,
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	HEEBI05	1047700	477	

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119	HEQAK71	598018	129	
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				L0663:1, S0406:1, L0743:1, L0754:1, L0750:1, L0780:1, L0581:1, L0603:1 and H0665:1.
	HEQCC55	884824	480	
	HEQCCSS	748227	481	
121	HERAR44	566811	131	AR060:18, AR055:17, AR299:10, AR089:10, AR283:10, AR185:9, AR104:8, AR039:8, AR096:7, AR282:6,
				AR316:6, AR277:6, AR176:5, AR300:5, AR204:5, AR162:5, AR161:5, AR163:5, AR181:4, AR233:4, AR201:4,
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			SEQ	
Gene No:	cDNA Clone	Contig ID:	HON XON	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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				AR258:1, AR296:1 H0059:1 and H0345:1.
122	HETBR16	703243	132	AR162:11, AR176:10, AR178:10, AR163:10, AR161:10, AR165:10, AR229:10, AR164:10, AR166:10, AR173:9,
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123	HFABH95	566712	133	AR173:16, AR162:14, AR161:14, AR163:13, AR180:12, AR178:11, AR257:11, AR262:11, AR191:11, AR196:10,
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				AR273:2, AR206:1, AR244:1, AR252:1 S6024:1, S0430:1, H0039:1, H0056:1 and H0660:1.

Gene No:	cDNA Clone Contig ID:	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
124	HFAEFS7	534142	134	AR291:14, AR161:14, AR162:13, AR163:13, AR313:10, AR242:10, AR201:10, AR165:9, AR164:9, AR252:9, AR197:9, AR194:9, AR053:9, AR196:9, AR198:8, AR241:14, AR197:9, AR194:9, AR053:9, AR196:9, AR198:8, AR245:8, AR245:8, AR194:9, AR204:7, AR204:6, AR204:6, AR207:7, AR191:7, AR204:6, AR212:6, AR204:6, AR204:6, AR204:6, AR212:6, AR204:6, AR204:5, AR190:5, AR204:5, AR204:5, AR190:5, AR204:5, AR204:5, AR190:5, AR204:6, AR204:6, AR204:6, AR204:4, AR204:2, AR204:2, AR204:2, AR204:1, AR204:1, AR204:1, AR204:1, AR204:2, AR204:2, AR204:2, AR204:1, AR204:2, AR204:2, AR204:1, AR204:2, AR204:1, AR204:2, AR204:1, AR204:2, AR204:2, AR204:2, AR204:2, AR204:2, AR204:2, AR204:1, AR204:2, AR204:1, AR204:2, AR20
125	HFAMB72	490697	135	AR262:10, AR161:8, AR162:8, AR163:8, AR198:7, AR180:5, AR176:5, AR055:5, AR060:5, AR223:5, AR165:5, AR309:5, AR164:5, AR179:5, AR290:5, AR166:5, AR089:4, AR300:4, AR171:4, AR104:4, AR183:4, AR242:4, AR263:4, AR245:4, AR245:3, AR269:3, AR269:3, AR269:3, AR269:3, AR269:3, AR269:3, AR269:3, AR269:3, AR269:3, AR277:3, AR168:3, AR277:3, AR216:3, AR290:3, AR299:3, AR299:1, AR299:1, AR299:1, AR188:1, AR299:1, AR398:1, AR299:1, L0799:1, L0779:1, L0777:1, L0777:1, and S0192:1.
126	HFCCQ50	579993	136	AR214:58, AR274:55, AR216:54, AR217:51, AR222:50, AR245:47, AR223:47, AR272:46, AR199:45, AR224:43, AR169:42, AR168:39, AR308:38, AR225:38, AR205:36, AR251:35, AR212:35, AR221:35, AR264:33, AR171:33, AR165:32, AR313:31, AR213:31, AR164:31, AR162:30, AR166:30, AR216:30, AR216:29, AR309:29, AR163:29, AR312:28, AR273:28, AR189:28, AR188:28, AR053:28, AR178:27, AR180:27, AR173:26, AR236:25, AR254:25, AR183:24, AR197:23, AR256:23, AR179:22, AR263:22, AR218:22, AR310:22, AR310:23, AR310:23, AR310:23, AR310:23, AR310:23, AR310:21, AR265:31, AR265:

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Gene No:	cDNA Clone ID	Contig ID:		Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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				AR296:13, AR060:12, AR196:12, AR289:12, AR289:12, AR239:12, AR229:12, AR198:12, AR198:12,
				AR177:12, AR204:11, AR185:11, AR287:11, AR237:11, AR295:11, AR231:11, AR244:10, AR192:10, AR248:10, AR238:10, AR280:9, AR286:9, AR315:9, AR104:9, AR285:9, AR249:9, AR226:9, AR294:9, AR235:8, AR234:8,
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127	HFCEB37	411345	137	AR282:18, AR176:14, AR269:13, AR183:11, AR173:11, AR201:11, AR182:11, AR252:11, AR204:11, AR193:11,
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				AR166:8, AR172:8, AR268:8, AR221:8, AR291:8, AR169:8, AR179:8, AR261:8, AR235:8, AR205:8, AR224:8,
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				AR229:7, AR192:7, AR255:7, AR258:7, AR191:6, AR254:6, AR096:6, AR177:6, AR055:6, AR195:6,
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				AR275:4, AR104:4, AR263:4, AR258:4, AR218:4, AR203:4, AR232:4, AR272:4, AR230:4, AR277:4, AR256:4,
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				S0134:1, S0045:1, H0747:1, H0013:1, H0009:1, S6028:1, L0598:1, L0532:1, S0052:1, H0696:1, S0146:1, L0439:1,
128	HEFAD50	\$20369	138	AR225:3. AR162:3. AR161:3. AR271:3. AR183:2. AR180:2. AR282:2. AR217:2. AR254:2. AR198:2. AR291:2.
77	Conveni	2020	}	AR175:2, AR288:2, AR177:2, AR201:2, AR163:2, AR267:2, AR224:2, AR295:2, AR266:2, AR312:2, AR173:2,
	_			AR277:2, AR311:2, AR238:2, AR033:2, AR193:2, AR228:2, AR294:2, AR195:2, AR275:1, AR243:1, AR272:1,
				AR205:1, AR174:1, AR213:1, AR293:1, AR308:1, AR229:1, AR233:1, AR285:1, AR247:1, AR269:1, AR181:1,
				AR182:1, AR230:1, AR296:1, AR185:1, AR240:1, AR297:1, AR258:1 H0172:2
129	HFGAD82	\$13669	139	
				ARU53:4, ARZ/4:4, ARU89:4, ARI92:4, ARZ/2:4, ARZ05:4, ARZ08:5, ART09:5, ARZ08:3, ARZ48:5, ARZ17:3, ART83:3, ARZ073:3, ART07:3, ARZ52:3, ARZ77:3, ARZ61:3, ARU39:3, ARZ45:3, ARZ176:3,
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				AR264:2, AR285:2, AR287:2, AR270:2, AR294:2, AR271:2, AR288:2, AR225:2, AR293:2, AR290:2, AR171:2,

Gene No:	cDNA Clone	Contig ID:	SEC Sec	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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				S0260:3, S0007.2, H0441:2, L3655:2, S0049:2, H0052:2, H0178:2, H0051:2, S6028:2, S0038:2, L0759:2, L0589:2,
				L0366:2, H0583:1, S0001:1, H0662:1, L3658:1, L0476:1, S0300:1, H0406:1, S6014:1, H0455:1, H0013:1, H0244:1,
				[H0390:1, S0346:1, H0327:1, H0041:1, H0563:1, H0567:1, S0050:1, S0048:1, S038:1, S0039:1, L0796:1, L5575:1, L04030:1, L0796:1, L0796:1, L0507:1, L0796:1, L0796:1, L0507:1, L0796:1, L0
				H0658:1, S0330:1, L0777:1, L0758:1, L0592:1 and L0593:1.
130	HFIIZ70	1043350	140	AR235:6, AR053:6, AR313:5, AR250:5, AR169:5, AR205:4, AR161:4, AR224:4, AR309:4, AR213:4, AR165:4,
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				AR060:2, AR188:2, AR191:2, AR240:2, AR295:2, AR297:2, AR173:2, AR217:2, AR236:2, AR316:2, AR294:2,
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				AR229:2, AR055:2, AR226:2, AR189:2, AR174:2, AR177:2, AR262:2, AR179:2, AR247:2, AR269:2, AR104:2,
				AR233:2, AR172:2, AR258:2, AR232:2, AR201:2, AR171:2, AR277:2, AR178:2, AR178:2, AR771:2,
_				AR203:2, AR210:2, AR190:2, AR182:2, AR168:1, AR228:1, AR288:1, AR234:1, AR231:1, AR222:1, AR199:1,
				AR239:1, AR033:1, AR266:1, AR268:1, AR216:1 H0617:16, H0545:12, L0757:8, S0358:1, S0360:1, L0747:1,
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				S0212:2, S0420:2, H0734:2, S0222:2, H0497:2, H0085:2, H0530:2, H0356:2, H0606:2, S0440:2, L0769:2, L0773:2,
				S0330.2, S0406.2, S3014.2, S0028.2, L0751.2, L0754.2, L0752.2, L0588.2, H0653.2, S0194.2, S0276.2, H0716.1,
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				[H0081:1, S0050:1, H0051:1, H0594:1, H0271:1, H0687:1, S0338:1, H0428:1, H0033:1, H0213:1, H0405:1, H0628:1,
				H0059:1, L0564:1, H0633:1, L0763:1, L3904:1, L0630:1, L0364:1, L0775:1, L0776:1, L0384:1, L5623:1, L2260:1,
				S0374:1, H0547:1, H0519:1, H0593:1, L3210:1, H0682:1, H0658:1, S0380:1, H0696:1, S0044:1, H0436:1, S0392:1,
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	HFIIZ/0	906/08	487	
131	HFIUR10	532060	141	AR169:4, AR165:4, AR161:3, AR163:3, AR215:3, AR162:3, AR166:3, AR246:3, AR252:3, AR313:3, AR089:3,
				AR311:3, AR266:2, AR270:2, AR180:2, AR261:2, AR164:2, AR269:2, AR096:2, AR286:2, AR289:2,
				AR201:2, AR297:2, AR312:2, AR205:2, AR217:2, AR1525:2, AR172:2, AR240:2, AR210:2, AR183:2, AR309:2, AR207:3, AP105:3, AP105:3, AP205:1, AP206:1, AP206:1, AP203:1, AP174:1, AP257:1
				ARI 15.2, ARZ91.2, ARI 10.2, ARI 20.2, ARZ52.1, ARZ07.1, ARZ07.1, ARZ27.1, ARX05.1, ARX17.1, ARZ51.1,

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Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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132	HFKET18	889515	142	AR213:6, AR180:6, AR242:5, AR162:5, AR165:5, AR178:4, AR309:4, AR165:4, AR257:4, AR272:4, AR166:4, AR229:3, AR292:4, AR183:4, AR300:4, AR296:3, AR296:2, AR296:2, AR297:2, AR297:3, AR297:2, AR297:3, AR2
	20 t V (T)	00/050		LU7/4:1, LU809:1, LU666:1, LU665:1, HU690:1, HU600:1, SU028:1, LU7:1:1, LU7:39:1, HU443:1, AU343:1, SU430:1, and H0352:1.
133	HFOXA73	850699	143	AR264:3, AR197:3, AR274:3, AR108:4, AR291:2, AR203:2, AR102:2, AR103:1, AR274:1, AR104:1, AR230:1, AR240:1, AR240:1, AR266:1, AR190:1, AR263:1, AR277:1, AR178:1, AR217:1, AR287:1, AR182:1, AR295:1 S0276:1
	HFOXA73	532079	483	
134	HFPA071	629193	144	AR061:490, AR273:461, AR232:455, AR237:432, AR238:424, AR227:414, AR226:343, AR241:311, AR186:304, AR274:285, AR244:270, AR206:269, AR194:260, AR192:197, AR271:181, AR243:173, AR052:167, AR198:167, AR274:285, AR244:270, AR206:269, AR194:260, AR192:191, AR271:181, AR243:173, AR265:167, AR204:152, AR310:151, AR292:150, AR205:148, AR259:147, AR229:136, AR312:132, AR213:132, AR213:132, AR213:123, AR213:123, AR233:128, AR248:123, AR249:122, AR246:99, AR246:99, AR234:98, AR096:96, AR218:96, AR280:93, AR309:93, AR306:92, AR295:10, AR282:104, AR246:99, AR243:198, AR096:96, AR218:66, AR286:34, AR315:83, AR179:80, AR263:17, AR256:74, AR267:72, AR247:71, AR316:66, AR295:66, AR298:65, AR284:64, AR183:63, AR296:36, AR283:37, AR203:37, AR203:37, AR266:31, AR291:24, AR176:11, AR161:9, AR162:9, AR163:9, AR245:7, AR245:7, AR285:7, AR181:7, AR235:7, AR235:7, AR182:7, AR183:8, AR181:7, AR235:7, AR236:7, AR165:7, AR178:7, AR245:7, AR285:7, AR181:7, AR236:7, AR285:7, AR183:8, AR181:7, AR236:7, AR256:7, AR165:7, AR178:7, AR245:7, AR181:7, AR236:7, AR180:7, AR256:7, AR193:8, AR181:7, AR236:7, AR256:7, AR165:7, AR178:7, AR245:7, AR180:7, AR245:7,

Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
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				AR210:4, AR189:4, AR311:4, AR230:4, AR172:4, AR188:4, AR203:4, AR199:3, AR190:3, AR211:3, AR260:2
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				5001615, H048612, H061515, H053512, 5042515, E004612, E0146612, E011512, E006013, E0061512, E0145013, E015413, F 0750-2 H0716-1 H0580-1 S0007-1 H0416-1 H0013-1 H0069-1 H0477-1 S0780-1 H0156-1 H0118-1 S0746-1
				H0581:1, T0103:1, H0050:1, L0471:1, H0014:1, S0214:1, H0328:1, H0628:1, H0135:1, H0551:1, S0440:1, L0662:1,
				L0794:1, L0650:1, L0775:1, L0805:1, L0776:1, L0655:1, L0606:1, L0783:1, L0809:1, L0792:1, S0374:1, H0693:1,
			İ	H0547:1, H0658:1, L0745:1, L0746:1, L0780:1, L0752:1, L0731:1, L0757:1, L0485:1 and H0422:1.
135	HFPCX09	1309793	145	AR252:67, AR253:31, AR251:7, AR104:5, AR180:5, AR273:5, AR161:4, AR265:4, AR243:4, AR249:3, AR309:3,
				AR052:3, AR282:3, AR313:3, AR060:3, AR172:3, AR184:3, AR245:3, AR193:3, AR089:3, AR283:3, AR163:3,
				AR165:3, AR033:3, AR207:3, AR202:3, AR299:3, AR213:3, AR312:3, AR162:3, AR164:2, AR310:2, AR200:2,
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				AR216:2, AR170:2, AR201:2, AR311:2, AR183:2, AR246:2, AR171:2, AR235:2, AR230:2, AR298:2, AR215:2,
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				AR237:2, AR295:2, AR290:2, AR266:2, AR268:2, AR277:2, AR231:2, AR270:2, AR285:1, AR263:1, AR267:1,
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				AR269:1, AR182:1, AR174:1, AR223:1, AR190:1, AR039:1, AR218:1, AR255:1, AR241::1, AR197:1, AR198:1,
				AR271:1, AR281:1, AR061:1, AR168:1, AR181:1, AR232:1, AR177:1, AR227:1, AR226:1, AR222:1, AR256:1,
				AR169:1, AR238:1, AR297:1, AR275:1, AR204:1, AR211:1 L0439:7, H0013:5, S0222:3, L0759:3, L0794:2, H0144:2, L0005:1, H0052:1, L0351:1 and L0742:1.
	HFPCX09	835390	484	
	HFPCX09	598723	485	
136	HFRAN90	520368	146	AR186:7, AR221:7, AR052:7, AR161:6, AR242:6, AR162:6, AR253:6, AR176:6, AR169:6, AR163:6, AR250:6,
				AR251:6, AR246:6, AR207:5, AR213:5, AR191:5, AR182:5, AR181:5, AR201:5, AR235:5, AR228:4, AR055:4,
				AR269:4, AR236:4, AR180:4, AR178:4, AR061:4, AR165:4, AR196:4, AR206:4, AR164:4, AR244:4, AR053:4,
				AR204:4, AR261:4, AR257:4, AR298:4, AR233:4, AR190:4, AR247:4, AR267:4, AR166:4, AR265:4, AR239:4,
				AR310:4, AR173:4, AR273:4, AR254:4, AR171:4, AR255:3, AR177:3, AR312:3, AR293:3, AR197:3, AR282:3,
				AR240:3, AR225:3, AR229:3, AR296:3, AR237:3, AR271:3, AR252:3, AR288:3, AR297:3, AR168:3, AR216:3,
				AR295:3, AR198:3, AR268:3, AR285:3, AR245:3, AR183:3, AR238:3, AR266:3, AR174:3, AR199:3, AR172:3,

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Gene	cDNA Clone	Contig ID:	ON X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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				AR311:1, AR219:1, AR039:1, AR217:1, AR222:1, AR194:1, AR170:1, AR241:1 S0050:1
137	HFTBM50	545012	147	AR300:4, AR104:4, AR240:4, AR277:3, AR060:3, AR185:3, AR055:3, AR299:2, AR316:2, AR282:2, AR219:2, AR089 AR089-2 AR283:2, AR218:2, AR096:2, AR039:2, AR313:1 L0439:6, L0731:4, L0769:2, L0666:2, S0432:2,
				\$0206.2, L0751.2, L0777.2, L0759.2, L0591.2, H0341.1, H0661.1, \$0408.1, H0601.1, H0497.1, H0123.1, L0471.1,
				H0051:1, H0252:1, H0673:1, H0616:1, H0551:1, H0646:1, S0422:1, L0372:1, L0771:1, L0773:1, L0768:1, L0775:1,
				L0375:1, L0527:1, L0664:1, L0665:1, S0374:1, H0519:1, H0659:1, H0521:1, H0522:1, L0747:1, L0749:1, L0755:1,
				L0758:1, S0031:1, L0683:1, L0590:1 and L0595:1.
138	HFTDL56	695976	148	AR202:8, AR244:8, AR192:7, AR289:7, AR270:7, AR266:6, AR198:6, AR246:6, AR186:6, AR274:6, AR243:6,
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				AR206:5, AR309:5, AR205:5, AR296:5, AR275:5, AR269:5, AR298:4, AR267:4, AR194:4, AR253:4, AR268:4,
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				AR283:3, AR271:3, AR249:3, AR310:3, AR213:3, AR104:3, AR229:3, AR184:3, AR313:3, AR294:3, AR232:3,
				AR240:3, AR185:3, AR300:3, AR316:3, AR175:2, AR089:2, AR177:2, AR293:2, AR299:2, AR248:2, AR096:2,
				AR039:2, AR226:2, AR231:2, AR227:2, AR183:2, AR237:2, AR241:2, AR233:2, AR285:2, AR234:2, AR258:2,
				AR259:1, AR263:1, AR219:1, AR218:1 H0024:38, H0123:15, H0208:5, H0209:1, H0617:1, H0264:1 and L0386:1.
139	HFTDZ36	545726	149	AR282.5, AR176.3, AR252.2, AR270.2, AR287.2, AR309.2, AR221.2, AR263.2, AR291.2, AR224.2, AR233.2,
				AR181:2, AR198:2, AR240:2, AR222:2, AR193:2, AR214:2, AR286:2, AR165:2, AR164:1, AR178:1, AR236:1,
				AR201:1, AR168:1, AR089:1, AR262:1, AR060:1, AR217:1, AR161:1, AR272:1, AR264:1, AR061:1, AR195:1,
				AR257:1, AR268:1, AR215:1, AR285:1, AR288:1, AR210:1, AR104:1, AR196:1 L0779:5, L0758:4, S0036:2,
				H0038:2, S0422:2, L0662:2, L0803:2, H0171:1, H0208:1, H0411:1, S0222:1, H0013:1, H0108:1, H0581:1, H0123:1,
				H0024:1, H0373:1, S0051:1, S6028:1, H0615:1, L0794:1, L0804:1, S0126:1, H0436:1, S0028:1, L0756:1, L0777:1,
				L0731:1 and S0242:1.
140	HFVIC62	799525	150	AR226:14, AR238:10, AR217:8, AR296:8, AR239:7, AR295:7, AR225:6, AR266:6, AR214:5, AR232:5, AR161:5,
				AR162:5, AR163:5, AR176:5, AR255:5, AR171:4, AR224:4, AR309:4, AR282:4, AR215:4, AR291:4, AR182:4,
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				AR183:3, AR261:3, AR221:3, AR311:3, AR178:3, AR297:3, AR294:3, AR267:3, AR286:3, AR231:3, AR316:3,
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Gene No:	cDNA Clone Contig ID:	Contig ID:	SEQ NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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141	HFXAM76	601402	151	AR221:3, AR168:3, AR242:3, AR180:3, AR161:3, AR163:2, AR060:2, AR195:2, AR172:2, AR264:2, AR039:2, AR309:2, AR379:2, AR277:2, AR275:1, AR265:1, AR282:1, AR266:1, AR183:1, AR270:1, AR162:1, AR165:1, AR212:1, AR212:1, AR210:1, AR193:1, AR193:1, AR266:1, AR183:1, AR270:1, AR162:1, AR212:1, AR246:1, AR283:1, H0457:9, H0081:2, H0083:2, H0628:2, L0761:2, L0800:2, H0658:2, H0762:2, H0254:2, H0255:2, H0255:2, H0253:2, H0253:2, H0081:2, H0083:2, H0581:1, H0052:1, R0360:1, H0722:1, R0441:1, H0561:1, H0069:1, H0427:1, H0657:1, H0082:1, R0366:1, H0087:1, H0364:1, H0488:1, L0435:1, H0080:1, H0560:1, R0427:1, H0488:1, L0435:1, H0080:1, H0560:1, R0427:1, L0663:1, L0663:1, L0663:1, L0663:1, L0663:1, L0663:1, H0670:1, H0670:1, H0670:1, H0670:1, H058:1, L0663:1, H051:1, R0445:1, S0440:1, L0662:1, R0670:1, H0670:1, H0670
142	HFXBL33	778070	152	AR163:25, AR161:24, AR162:24, AR313:23, AR173:17, AR180:17, AR196:17, AR166:16, AR229:16, AR164:16, AR270:14, AR270:14, AR182:14, AR238:14, AR234:14, AR175:14, AR179:13, AR269:13, AR181:13, AR179:12, AR285:12, AR262:12, AR240:11, AR233:11, AR257:11, AR183:11, AR264:11, AR300:10, AR268:10, AR285:10, AR285:10, AR287:10, AR287:10, AR297:10, AR297:10, AR286:10, AR286:10, AR287:10, AR297:10, AR287:10, AR297:10, AR297:10, AR287:10, AR297:10, AR297:10, AR287:10, AR297:10, AR297:
143	HFXGT26	745381	153	AR254:6, AR180:6, AR215:6, AR165:5, AR166:5, AR164:5, AR269:5, AR178:5, AR162:5, AR161:4, AR176:4, AR282:4, AR163:4, AR275:4, AR270:4, AR089:4, AR253:4, AR235:4, AR252:4, AR198:4, AR204:4, AR183:4,

Gene	cDNA Clone	Contig ID:	SEQ 15 NO.Y	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
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				AR316:2, AR289:2, AR274:2, AR201:2, AR207:2, AR233:2, AR297:2, AR205:2, AR236:2, AR293:2, AR263:2,
				AR237.2, AR308.2, AR216.2, AR294.2, AR288.2, AR271.2, AR239.2, AR053.2, AR246.2, AR258.2, AR227.2,
				AR177:2, AR290:2, AR212:2, AR214:2, AR174:2, AR243:2, AR061:2, AR232:2, AR287:1, AR104:1, AR195:1,
				AR169:1, AR199:1, AR033:1, AR190:1, AR285:1, AR311:1, AR231:1, AR291:1, AR277:1, AR196:1, AR185:1,
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				H0615:2, S0052:2, L0589:2, L0599:2, H0556:1, S0114:1, S0001:1, H0306:1, S0360:1, H0392:1, H0632:1, T0082:1,
				H0318:1, H0581:1, H0154:1, H0634:1, S0428:1, H0519:1, S0152:1, L0779:1, S0031:1, H0343:1 and L0596:1.
144	HFXJU68	1352218	154	AR182.8, AR176.8, AR309:7, AR228:7, AR269:7, AR267:6, AR229:6, AR268:6, AR181:6, AR266:6, AR178:6,
:				AR233:6, AR197:6, AR270:6, AR180:6, AR201:6, AR162:6, AR161:6, AR163:5, AR168:5, AR204:5, AR257:5,
				AR261:5, AR177:5, AR207:5, AR165:5, AR193:5, AR236:5, AR271:5, AR293:5, AR238:5, AR164:5, AR239:5,
				AR225.5, AR166.5, AR061.5, AR237.5, AR289.5, AR226.5, AR055.5, AR183.5, AR291.4, AR060.4, AR255.4,
			<u> </u>	AR089:4, AR224:4, AR179:4, AR175:4, AR296:4, AR214:4, AR264:4, AR231:4, AR300:4, AR262:4, AR286:4,
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				[AR198:3, AR290:3, AR039:3, AR173:3, AR299:3, AR242:3, AR294:3, AR096:3, AR295:3, AR234:3, AR033:3,
				AR222:3, AR216:3, AR213:3, AR235:3, AR191:3, AR288:3, AR196:3, AR205:3, AR174:3, AR282:3, AR185:3,
				AR192:3, AR195:3, AR232:3, AR240:3, AR190:3, AR283:3, AR285:3, AR246:3, AR203:2, AR172:2, AR277:2,
				AR189:2, AR308:2, AR297:2, AR188:2, AR199:2, AR200:2, AR104:2, AR311:2, AR258:2, AR171:2, AR256:2,
				AR223:2, AR212:2, AR210:2, AR260:2, AR211:2, AR275:2, AR313:2, AR221:1, AR219:1, AR245:1 S0282:1,
	HFXJU68	570855	486	
145	HFXIX44	701988	155	1
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				AR233:7, AR226:7, AR309:7, AR247:7, AR192:7, AR239:7, AR180:7, AR236:7, AR257:7, AR293:7, AR266:7,
				[AR235:7, AR240:7, AR238:7, AR267:7, AR096:7, AR177:7, AR261:6, AR053:6, AR179:6, AR245:6, AR268:6,
				AR282:6, AR299:6, AR198:6, AR290:6, AR204:6, AR191:6, AR060:6, AR262:6, AR174:6, AR277:6, AR312:6,
				AR271:6, AR185:5, AR316:5, AR289:5, AR270:5, AR294:5, AR193:5, AR201:5, AR258:5, AR296:5, AR212:5,
				AR237:5, AR255:5, AR227:5, AR234:5, AR061:5, AR274:5, AR275:5, AR264:5, AR197:5, AR287:5, AR243:5,
				AR297.5, AR286:4, AR263:4, AR199:4, AR200:4, AR231:4, AR203:4, AR291:4, AR214:4, AR242:4, AR285:4,
				AR230:4, AR033:4, AR189:4, AR213:4, AR188:4, AR195:4, AR288:4, AR246:4, AR295:4, AR224:4, AR252:3,
			į	AR104:3, AR250:3, AR272:3, AR218:3, AR219:3, AR190:3, AR308:3, AR222:3, AR171:3, AR260:3, AR207:3,

Gene No:	cDNA Clone ID	Contig ID:	SEQ. ID NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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146	HFXKT05	069859	l l	AR207:65, AR197:54, AR193:47, AR192:45, AR201:42, AR033:40, AR299:40, AR055:39, AR242:38, AR235:38, AR177:38, AR233:37, AR198:35, AR185:33, AR060:33, AR195:32, AR174:31, AR203:31, AR191:31, AR204:31, AR204:31, AR204:31, AR204:31, AR204:31, AR204:32, AR177:38, AR233:37, AR198:35, AR187:39, AR245:28, AR196:28, AR196:28, AR196:28, AR196:28, AR196:28, AR196:28, AR196:28, AR196:28, AR204:31, AR291:27, AR291:21, AR291:31, AR291:1, AR291:31, AR291:3
147	HGBFO79	422794	157	AR207:46, AR217:35, AR263:35, AR214:34, AR216:31, AR311:31, AR223:31, AR235:30, AR264:30, AR222:30, AR192:27, AR309:26, AR170:26, AR266:26, AR168:25, AR169:25, AR224:24, AR308:24, AR225:23, AR192:27, AR309:26, AR170:26, AR266:26, AR168:25, AR169:21, AR162:21, AR277:21, AR165:20, AR291:20, AR64:20, AR261:19, AR312:19, AR245:19, AR161:10, AR172:19, AR163:18, AR289:18, AR291:20, AR164:20, AR261:19, AR312:19, AR245:19, AR166:17, AR246:17, AR246:17, AR236:17, AR201:16, AR288:16, AR272:18, AR272:16, AR282:15, AR282:15, AR282:16, AR272:14, AR272:14, AR272:14, AR272:13, AR262:15, AR262:15, AR262:13, AR262:12, AR18:12, AR300:12, AR300:12, AR300:12, AR272:13, AR262:13, AR262:13, AR262:13, AR262:13, AR262:13, AR262:11, AR300:12, AR300:12, AR300:12, AR300:12, AR316:13, AR262:10, AR282:10, AR293:10, AR282:10, AR282:10, AR293:10, AR293:10, AR293:10, AR293:10, AR293:10, AR293:12, L0749:5, H0556:4, AR230:8, AR2

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148	HGBHI35	570262	158	AR089.24, AR226.21, AR299.20, AR164.20, AR165.20, AR060.19, AR166.17, AR185.16, AR201.16, AR163.15,
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149	HGBIB74	837220	159	AR214:16, AR216:13, AR217:11, AR215:9, AR161:9, AR162:9, AR163:9, AR176:8, AR250:8, AR165:8, AR178:7,
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	HGBIB74	899864	488	
150	HGLAF75	566838	160	
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151	HHEMA75	494099	161	AR245:10, AR207:7, AR197:7, AR242:6, AR169:6, AR282:6, AR221:6, AR243:6, AR195:5, AR224:5, AR309:5,
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152	HHENK42	493724	162	AR180:6, AR165:5, AR245:5, AR164:5, AR204:5, AR039:5, AR166:5, AR313:5, AR216:5, AR242:5, AR183:4, AR089:4, AR275:4, AR178:4, AR173:4, AR163:4, AR175:4, AR096:4, AR269:4, AR162:3, AR201:3, AR181:3,
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153	HHENV10	562772	163	AR242:3, AR235:3, AR183:3, AR309:3, AR282:3, AR243:2, AR171:2, AR283:1, AR055:1, AR257:1, AR168:1, AR213:1, AR164:1, AR230:1, AR264:1, AR287:1 H0543:2, H0497:1 and H0625:1.
154	HHEPM33	877639	162	AR263:38, AR207:37, AR311:31, AR264:30, AR212:29, AR195:27, AR309:27, AR308:26, AR165:26, AR164:25,
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156	HHGCG53	340818	166	AR192:3, AR169:3, AR264:3, AR162:3, AR309:3, AR245:3, AR250:3, AR161:3, AR163:3, AR171:3, AR193:2, AR266:2, AR176:2, AR289:2, AR289:2, AR267:2, AR275:2, AR239:2, AR295:2, AR238:2, AR225:2, AR182:2, AR182:2, AR263:2, AR263:2, AR183:2, AR183:2, AR183:2, AR183:2, AR183:2, AR269:1, AR263:1, AR263:1, AR263:1, AR263:1, AR263:1, AR290:1, AR290:1, AR240:1, AR229:1, AR282:1, AR280:1, AR290:1, AR2
157	HHGCM76	662329	167	AR245;8, AR175:7, AR183:6, AR176:6, AR196:6, AR191:6, AR174:6, AR060:5, AR254:5, AR263:5, AR039:5, AR173:5, AR177:7, AR309:5, AR261:5, AR232:4, AR161:4, AR162:4, AR162:4, AR163:4, AR182:4, AR264:4, AR173:5, AR177:5, AR198:4, AR261:5, AR270:4, AR268:4, AR168:4, AR189:4, AR166:3, AR242:3, AR242:1, AR242:3, AR242:3, AR242:2, AR242:2, AR242:3, AR242:3, AR242:2, AR242:3, AR242:3, AR242:3, AR242:2, AR242:3, AR242:3, AR242:2, AR242:3, AR242:2, AR242:2, AR242:3, AR242:3, AR242:2, AR242:2, AR242:3, AR242:3, AR242:2, AR242:2, AR242:3, AR242:3, AR242:2, AR242:2, AR242:2, AR242:3, AR242:3, AR242:2, AR242:2, AR242:3, AR242:2, AR242:2, AR242:2, AR242:3, AR242:2, AR242:2, AR242:3, AR242:3, AR242:2, AR242:2, AR242:3, AR242:3, AR242:2, AR242:2, AR242:3, AR242:3, AR242:2, AR242:2, AR242:3, AR242:3, AR242:3, AR242:3, AR242:2, AR242:2, AR242:3, AR242:3, AR242:3, AR242:2, AR242:3, AR242:2, AR242:2, AR242:2, AR242:3, AR242:2, AR2

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158	HHGDW43	554613	168	AR161:7, AR163:7, AR176:7, AR266:7, AR182:6, AR165:6, AR178:6, AR253:6, AR233:6, AR233:6, AR265:6, AR263:6, AR263:6, AR265:5, AR266:6, AR166:6, AR268:5, AR181:5, AR269:5, AR267:5, AR229:5, AR309:5, AR177:5, AR285:5, AR257:5, AR228:5, AR183:4, AR063:4, AR197:4, AR061:4, AR231:34, AR272:4, AR261:4, AR089:4, AR174:4, AR231:4, AR230:4, AR230:4, AR296:4, AR104:4, AR271:4,
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159	HHPEC09	695726	169	AR254:11, AR309:9, AR264:8, AR253:8, AR176:8, AR173:7, AR182:7, AR169:7, AR268:7, AR269:7, AR162:7,
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162	HJABB94	456466	172	AR176:9, AR225:9, AR221:8, AR295:8, AR170:8, AR264:8, AR178:8, AR288:7, AR291:7, AR180:7, AR215:7,
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163	HJABX32	487807	173	AR060:16, AR055:15, AR271:11, AR282:10, AR104:10, AR089:9, AR283:9, AR299:8, AR253:7, AR185:7,
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164	HJACG30	895505	174	AR263:8, AR165:8, AR250:8, AR162:7, AR161:7, AR205:7, AR196:7, AR166:7, AR164:7, AR215:7, AR163:7,
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	HJACG30	774300	491	
165	HJBCY35	719729	175	AR215:11, AR291:11, AR225:10, AR217:9, AR216:8, AR296:8, AR214:8, AR297:8, AR266:7, AR183:7, AR257:7,
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169	HKABZ65	862030	179	AR313:41, AR242:32, AR039:28, AR165:25, AR163:25, AR164:24, AR161:24, AR162:24, AR166:24, AR089:24, AR096:23, AR173:22, AR196:20, AR193:20, AR299:20, AR300:20, AR258:20, AR180:19, AR175:19, AR178:18, AR240:18, AR229:18, AR234:18, AR185:17, AR241:17, AR218:17, AR262:17, AR179:16, AR285:16, AR183:16, AR269:16, AR293:15, AR179:15, AR182:15, AR181:15, AR293:15, AR179:15, AR199:15, AR182:15, AR181:13, AR264:12, AR296:14, AR296:14, AR270:14, AR270:14, AR270:13, AR270:13, AR271:13, AR264:12, AR200:12, AR230:11, AR195:12, AR281:1, AR260:11, AR200:11, AR200:11, AR281:11, AR189:11, AR260:10, AR281:10, AR188:10, AR201:10, AR188:10, AR201:10, AR188:10, AR201:10, AR188:10, AR201:10, AR189:11, AR261:13, AR201:13, AR201:1
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170	HKACB56	554616	180	
171	HKACD58	1352202	181	AR261:30, AR235:29, AR283:29, AR297:20, AR291:17, AR285:16, AR286:15, AR295:13, AR183:13, AR269:13, AR287:12, AR258:11, AR268:11, AR266:11, AR289:10, AR161:10, AR162:10, AR288:10, AR216:10, AR288:10, AR288:10, AR266:10, AR165:10, AR163:10, AR166:9, AR207:9, AR270:9, AR282:9, AR277:8, AR223:8, AR214:8, AR243:8, AR215:8, AR224:8, AR224:8, AR096:8, AR039:8, AR172:8, AR221:8, AR192:8, AR182:8, AR104:8,

Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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172	НКАDQ91	604123	182	AR211:37, AR199:28, AR275:8, AR215:8, AR210:7, AR245:7, AR234:6, AR238:6, AR239:5, AR224:5, AR178:5,
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173	HKAEV06	1352263	183	AR272:35, AR165:34, AR163:33, AR164:33, AR161:32, AR162:32, AR245:32, AR166:32, AR274:28, AR212:28,
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174	HKAFK41	545018	184	
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175	HKAFT66	946512	185	AR214:32, AR195:28, AR222:28, AR169:27, AR223:26, AR224:25, AR168:23, AR172:23, AR235:22, AR217:21, AR211:20, AR216:20, AR222:28, AR169:21, AR221:19, AR171:18, AR263:18, AR225:17, AR264:16, AR215:15, AR281:15, AR196:14, AR170:14, AR212:14, AR261:13, AR262:13, AR263:12, AR262:12, AR309:12, AR211:11, AR165:11, AR166:11, AR265:11, AR164:11, AR161:11, AR308:11, AR315:11, AR210:10, AR254:10, AR291:11, AR265:11, AR266:11, AR265:11, AR199:11, AR308:11, AR315:11, AR210:10, AR254:10, AR291:11, AR262:10, AR200:8, AR210:10, AR297:9, AR093:9, AR280:8, AR280:8, AR272:7, AR251:7, AR261:7, AR262:7, AR262:7, AR177:7, AR314:7, AR312:7, AR310:8, AR280:6, AR287:8, AR287:6, AR280:6, AR280:4, AR280:4, AR280:4, AR280:4, AR280:4, AR280:4, AR280:3, AR280:
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	HKAFT66	904790	496	
176	HKB1E57	876571	186	AR253:4, AR225:3, AR171:3, AR205:3, AR192:3, AR169:3, AR245:2, AR282:2, AR193:2, AR274:2, AR039:2, AR291:2, AR212:2, AR163:2, AR162:2, AR266:2, AR161:2, AR269:2, AR264:1, AR271:1, AR178:1, AR316:1, AR291:2, AR261:1, AR168:1, AR270:1, AR183:1, AR297:1, AR283:1 L0747:4, L0766:3, L0776:3, L0665:3, H0328:2, L0763:2, L0763:2, L0769:2, L0772:2, L0764:2, L0666:2, L0745:2, L0750:2, L0777:2, L0769:2, H0658:1, H0658:1, H0673:1, H0619:1, H0492:1, H0156:1, H0421:1, H0620:1, S0051:1, H0083:1, H0510:1, H0266:1, H0631:1, H0649:1, L0774:1, L0806:1, L0807:1, H0144:1, H0690:1, H0658:1, H0521:1, H0522:1, L0774:1, L0774:1, L0774:1, H0543:1 and H0422:1.
	HKB1E57	654871	497	
177	HKFBC53	1352286	187	AR249:155, AR248:131, AR251:111, AR265:54, AR253:42, AR096:23, AR263:23, AR244:18, AR290:13, AR268:13, AR246:12, AR184:11, AR177:11, AR194:9, AR267:8, AR270:8, AR247:7, AR240:7, AR269:7, AR183:6, AR202:5, AR175:5, AR234:5, AR241:5, AR316:5, AR206:5, AR313:5, AR055:4, AR299:4, AR033:4, AR238:3, AR292:3, AR061:3, AR182:3, AR171:3, AR273:3, AR274:3, AR198:3, AR275:3, AR216:3, AR266:3, AR195:3, AR284:3, AR168:3, AR237:2, AR215:2, AR282:2, AR282:2, AR310:2, AR350:2, AR300:2,

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	HKFBC53	513190	499	
	HKFBC53	383426	200	
178	HKGC027	601969	188	AR170:6, AR282:6, AR235:5, AR180:5, AR215:5, AR225:4, AR263:4, AR053:4, AR271:4, AR161:4, AR162:4,
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180	HKISB57	625956	061	AR161:12, AR162:12, AR163:12, AR165:12, AR164:11, AR166:11, AR089:8, AR225:7, AR178:6, AR183:6, AR172:6, AR300:5, AR224:5, AR181:5, AR221:5, AR223:5, AR170:5, AR299:5, AR039:4, AR291:4, AR096:4, AR268:4, AR275:4, AR286:4, AR274:4, AR274:4, AR277:4, AR277:4, AR276:4, AR269:3, AR276:3, AR179:3, AR240:3, AR173:3, AR173:3, AR175:3, AR240:3, AR240:3, AR173:3, AR175:3, AR175:3, AR240:3, AR240:3, AR240:3, AR270:3, AR270:
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186	HLDOW79	847396	196	AR252:214, AR264:119, AR250:104, AR254:94, AR311:91, AR194:85, AR308:83, AR202:81, AR195:78, AR263:76,	
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cDNA Clone ID		HLDQR62	нг.роси79
Gene No:		187	188

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205	HIMADK33	561941	215	AR283:32, AR096:20, AR089:18, AR218:17, AR104:17, AR277:16, AR039:16, AR316:15, AR282:15, AR055:13,
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				LOS15:1, L0809:1, S0428:1, S0216:1, H0699:1, H0695:1, H0684:1, H0648:1, H0710:1, H0241:1, H0696:1, H01436:1, H0780:1, L0750:1, L0779:1, L0731:1, S0260:1, H0595:1, L0599:1, S0192:1, S0276:1, H0542:1 and
				H0352:1,
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				AR290:1, AR195:1, AR288:1, AR267:1 H0521:2, S0436:2, S0358:1, S0360:1, H0266:1, S0144:1, L0646:1, L0655:1, L0791:1, L0666:1, H0435:1, H0660:1, S0152:1 and H0665:1.
	HMADU73	467053	206	
207	HMAMI15	1352406	217	AR060:14, AR283:13, AR055:10, AR277:9, AR282:9, AR185:9, AR104:9, AR300:8, AR096:8, AR316:8, AR299:8,
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				and S0462:1.
	HMAMI15	1049263	202	
708	HMCFY13	635301	218	AR176:8, AR161:6, AR162:6, AR266:6, AR181:6, AR269:6, AR163:6, AR172:6, AR228:5, AR267:5, AR233:5, AR055:5, AR268:5, AR229:5, AR165:5, AR309:5, AR238:4, AR183:4, AR178:4, AR164:4, AR237:4, AR215:4,
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Gene	cDNA Clone		SEQ ID	Tissue Distribution Library Code: Count
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209	HMDAB56	9/9095	612	AR168:4, AR161:4, AR162:4, AR212:4, AR163:4, AR222:4, AR216:4, AR172:4, AR264:3, AR214:3, AR289:3, AR111:3, AR110:3, AR210:3, AR250:3, AR271:3, AR225:3, AR299:3, AR165:3, AR164:3, AR164:3,
				AR171:2, AR253:2, AR096:2, AR199:2, AR201:2, AR308:2, AR221:2, AR263:2, AR039:2, AR312:2, AR205:2,
				AR196.2, AR294.2, AR213.2, AR267.2, AR217.2, AR290.2, AR274.2, AR166.2, AR291.2, AR295.2, AR089.2,
				AR193:2, AR191:1, AR316:1, AR033:1, AR240:1, AR269:1, AR215:1, AR266:1, AR224:1, AR195:1, AR293:1,
				AR283:1, AR183:1, AR189:1, AR262:1, AR104:1, AR210:1, AR247:1, AR239:1, AR268:1, AR169:1 L0809:2,
				H0346:1, H0271:1, L0774:1 and L0532:1.
210	HMDA029	600406	220	AR313:15, AR196:15, AR175:13, AR179:12, AR242:12, AR161:11, AR162:11, AR262:11, AR207:11, AR229:11,
ì				AR163:11, AR192:10, AR181:10, AR178:10, AR173:10, AR053:10, AR191:10, AR165:10, AR234:10, AR257:10,
				AR299:10, AR164:10, AR233:10, AR166:9, AR180:9, AR236:9, AR174:9, AR238:9, AR258:9, AR247:9, AR231:9,
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				[AR243:7, AR189:7, AR287:7, AR210:7, AR294:7, AR230:7, AR267:7, AR197:7, AR255:6, AR212:6, AR297:6,
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				AR033:5, AR193:5, AR216:5, AR275:5, AR168:5, AR282:5, AR250:5, AR225:5, AR312:5, AR286:5, AR256:5,
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				AR060:4, AR215:4, AR096:4, AR218:4, AR308:4, AR253:4, AR274:4, AR277:4, AR316:4, AR219:4, AR252:4,
				AR214:3, AR169:3, AR224:3, AR289:3, AR061:3, AR245:3, AR232:3, AR210:3, AR309:3, AR195:3, AR055:3,
				AR211:3, AR311:3, AR221:3, AR039:3, AR172:2, AR217:2, AR104:2, AR283:1 H0346:1 and H0553:1.
211	HMECK83	636035	221	AR313:19, AR165:17, AR164:17, AR166:16, AR161:14, AR163:13, AR162:13, AR183:13, AR216:13, AR173:13,
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5	UNCEPTIO	366033	222	ARZILIS, ARZZIS, ARZZZS, ARIOS. 110200.1 ARZZZZZ ARIOSIS, 27 ARZZZZZ ARZZZZZZZZZZZZZZZZZZZZZZZZZZZZ
717	HIMEED 18	2001/13	777	ALCOLOT, ANTONOS, ALCOLOS, ANTONOS, ANT

Gene	cDNA Clone	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
213	HMEFT54	520307	223	AR060:7, AR055:7, AR039:6, AR222:5, AR196:5, AR089:5, AR104:5, AR269:5, AR176:5, AR161:5, AR162:5, AR182:5, AR240:5, AR240:5, AR263:5, AR265:5, AR295:5, AR240:5, AR309:5, AR204:5, AR182:4, AR243:4, AR243:4, AR246:4, AR316:4, AR166:4, AR300:4, AR247:4, AR247:4, AR369:5, AR313:4, AR228:4, AR243:4, AR229:4, AR255:4, AR274:4, AR266:4, AR266:4, AR283:4, AR247:4, AR290:4, AR236:4, AR261:4, AR294:4, AR295:3, AR192:3, AR275:3, AR176:3, AR176:3, AR275:3, AR265:3, AR265:3, AR199:3, AR197:3, AR275:3, AR237:3, AR265:3, AR193:3, AR178:3, AR265:3, AR265:3, AR193:3, AR266:3, AR286:3, AR286:3, AR286:3, AR191:3, AR239:3, AR171:3, AR266:3, AR290:3, AR287:3, AR286:3, AR287:3, AR286:3, AR191:3, AR214:2, AR286:3, AR290:2, AR290:3, AR191:3, AR214:2, AR286:3, AR290:2, AR290:2, AR290:2, AR290:2, AR290:3, AR198:3, AR200:3, AR290:3, AR290:2, AR290:2, AR290:2, AR290:2, AR290:2, AR290:1, L0055:1, L0055:1, L0055:1, L0055:1, L0055:1, L0075:1, L0071:1, L0071:1, L0071:1, and

PCT/US02/08277

Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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215	HMIAL37	603201	225	AR265:6, AR207:6, AR176:6, AR217:5, AR162:5, AR161:5, AR225:5, AR183:5, AR183:5, AR182:5, AR269:5, AR245:5, AR223:5, AR214:4, AR281:4, AR216:4, AR216:4,
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216	HMKCG09	2480/8	977	AR202:39, AR292:23, AR280:23, AR313:23, AR104:24, AR310:24, AR284:23, AR312:20, AR032:20, AR283:13, AR28314:19 AR314:19 AR309:19 AR275:19. AR266:18. AR186:18. AR033:17. AR060:17. AR295:17. AR285:17, AR283:16,
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_				L07/0:1, L0804:1, L0/88:1, H0/30:1, L0/43:1, L0/30:1, L0/31:1 and L0301:1.
217	HMMAH60	562776	227	AR242:10, AR313:9, AR192:9, AR196:7, AR173:7, AR165:7, AR089:7, AR164:6, AR197:6, AR039:6, AR161:6, AD163:6, AB103:5, AD103:5, AD100:5, AD100:5, AD103:5, AD103:5, AD100:5, AD100:5, AD103:5, AD103:5, AD100:4, AD100:5, AD100:4, AD
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				AR225:2, AR232:2, AR215:2, AR217:1, AR253:1, AR055:1, AR061:1 L0547:1 and H0444:1.
218	HMQDT36	1309723	228	AR218:25, AR219:21, AR096:17, AR039:13, AR316:13, AR089:12, AR299:9, AR055:9, AR282:9, AR060:8,
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lone C		\vdash						┝
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Gene No:			219				220	CCC

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Gene No.	cDNA Clone	Contig ID:	SEQ ID	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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226	HMWDC28	460487	236	AR245:5, AR176:5, AR198:5, AR161:5, AR162:4, AR204:4, AR163:4, AR207:4, AR271:4, AR309:4, AR266:4, AR164:4, AR165:4, AR166:4, AR181:3, AR221:3, AR039:3, AR252:3, AR089:3, AR254:3, AR216:3, AR182:3, AR291:3, AR177:3, AR257:3, AR224:3, AR264:3, AR312:3, AR268:3, AR275:3, AR275:3, AR296:3, AR178:2, AR177:3, AR257:2, AR267:2, AR196:2, AR295:2, AR311:2, AR055:2, AR233:2, AR282:2, AR270:2, AR288:2, AR269:2, AR191:2, AR269:2, AR185:2, AR300:2, AR285:2, AR286:2, AR286:2, AR234:2, AR286:2, AR269:2, AR316:2, AR316:2, AR316:2, AR265:2, AR231:2, AR300:2, AR286:2, AR294:2, AR294:2, AR297:2, AR297:2, AR297:2, AR292:2, AR292:1, L0655:1, L0756:1, L0757:1 and L0591:1.
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228	HMWFY10	825421	238	AR176:6, AR161:6, AR162:5, AR163:5, AR181:5, AR055:5, AR269:5, AR229:5, AR060:5, AR204:5, AR228:5,
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229	HMWGY65	1308287	239	
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233	HNFIY77	634551	243	AR241:9, AR313:8, AR194:8, AR186:7, AR192:7, AR202:7, AR206:7, AR161:7, AR162:7, AR163:6, AR204:6, AR204:6, AR229:6, AR186:7, AR192:7, AR238:6, AR166:5, AR207:5, AR198:5, AR225:5, AR208:5, AR204:6, AR207:5, AR198:5, AR207:5, AR198:5, AR207:5, AR198:5, AR207:5, AR198:5, AR207:4, AR207:3, AR207:1, AR207:1, AR207:1, AR207:1, H0619:1, H0622:1, H0621:1, H0622:1, H0613:1, H0621:1, H06
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235	HNFJH45	410107	245	AR176:7, AR266:6, AR235:6, AR267:6, AR060:5, AR228:5, AR229:5, AR178:5, AR269:5, AR055:4, AR239:4, AR161:4, AR182:4, AR183:4, AR163:4, AR163:4, AR163:4, AR162:4, AR182:4, AR182:4, AR182:4, AR182:4, AR162:4, AR226:4, AR270:3, AR230:3, AR201:3, AR296:3, AR270:3, AR291:3, AR297:3, AR298:3, AR175:3, AR231:3, AR173:3, AR173:3, AR165:3, AR165:3, AR288:3, AR287:3, AR288:3, AR287:3, AR287:3, AR294:3, AR296:3, AR296:3, AR288:2, AR286:2, AR286:2, AR247:2, AR089:2,

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e cDNA Clone ID		HNGIH43								HNGIJ31							-		HNGIQ46								
Gene No:		239		_		_				240	: 								241								

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243	HNGJP69	604891	253	AR313:28, AR162:20, AR161:20, AR163:19, AR165:18, AR164:17, AR166:17, AR089:16, AR173:14, AR242:14, AR096:14, AR299:12, AR247:12, AR300:11, AR178:11, AR258:11, AR193:11, AR175:10, AR240:10, AR262:10,
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	HNG0112	839283	516	
247	HNHAH01	496115		AR198:6, AR309:6, AR161:6, AR162:6, AR163:6, AR266:6, AR181:5, AR228:5, AR176:5, AR267:5, AR183:5, AR201:5, AR233:5, AR236:5, AR165:4, AR177:4, AR269:4, AR182:4, AR164:4, AR204:4, AR261:4, AR229:4,

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	HNHEI42	842223	518	
	HNHE142	823723	519	
249	HINHEU93	634851	259	AR313:24, AR173:20, AR162:16, AR161:16, AR163:16, AR165:15, AR247:14, AR164:14, AR166:14, AR175:13, AR258:13, AR242:13, AR292:10, AR292:10, AR292:10, AR290:10, AR240:9, AR258:13, AR293:12, AR254:9, AR264:9, AR180:9, AR196:9, AR179:9, AR312:9, AR199:9,
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250	HINHEM14	664507	260	I

Gene No:	cDNA Clone	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
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				H0052:1, H0570:1, H0012:1, H0014:1, H0510:1, H028:1, H0622:1, S0366:1, H0040:1, H0623:1, L0351:1, T0042:1, h0561:1 t0764:1 t0767:1 t0805:1 t0809:1 S0053:1 t13828:1 H0520:1 H0435:1 H0659:1 S3014:1
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251	HNHNB29	895462	261	AR313:23, AR254:22, AR162:20, AR161:20, AR163:19, AR173:17, AR165:16, AR164:16, AR166:15, AR229:14,
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252	HNHOD46	843488	262	
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				AR1/2:1, AR21:1, AR260:1, AR253:1, AR061:1 S0216:1

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253		834927	263	
254	HNTB126	1310821	564	AR195:19, AR214:19, AR194:18, AR225:16, AR223:16, AR164:16, AR165:15, AR281:16, AR196:15, AR224:15, AR195:19, AR214:19, AR194:18, AR225:16, AR164:14, AR195:14, AR215:14, AR215:15, AR215:14, AR195:14, AR244:14, AR169:14, AR167:11, AR264:14, AR169:14, AR273:11, AR264:14, AR244:14, AR169:14, AR268:11, AR265:11, AR265:11, AR267:3, AR267:3, AR295:3, AR295:3, AR295:3, AR295:3, AR295:11, AR295:10, AR245:10, AR265:10, AR267:3, AR296:3,
	HNTB126	796807	520	
	HNTB126	590738	521	

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				L0749:1, L0779:1, L0777:1, L0755:1 and L0731:1.
257	HNTCE26	1160395	267	AR291:7, AR164:5, AR295:5, AR296:5, AR285:5, AR166:5, AR165:5, AR170:4, AR297:4, AR287:4, AR162:4,
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Gene No:	Gene cDNA Clone	Contig ID:	SEQ D No:X	
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258	HNTNI01	1352285	268	AR207:15, AR263:12, AR169:11, AR311:11, AR212:10, AR198:10, AR264:10, AR235:10, AR252:9, AR168:9, AR2223:9, AR224:9, AR089:9, AR053:8, AR215:8, AR172:8, AR161:8, AR162:8, AR214:8, AR222:8, AR163:8,
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	HNTNI01	699848	523	
259	HODDF13	684307	569	AR312:21, AR308:20, AR205:19, AR253:19, AR250:19, AR309:19, AR264:18, AR311:16, AR212:16, AR213:15,
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Gene	cDNA Clone Contig ID:	Contig ID:	SEQ ID	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
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261	HODDN92	422913	271	AR161:4, AR162:4, AR192:4, AR192:4, AR308:4, AR264:4, AR176:4, AR311:3, AR164:3, AR309:3, AR166:3, AR312:3, AR213:3, AR214:3, AR192:3, AR225:3, AR313:3, AR089:3, AR270:3, AR270:3, AR172:3, AR235:3, AR289:2, AR281:2, AR291:2, AR281:2, AR282:2, AR294:2, AR185:2, AR282:2, AR290:2, AR290:2, AR290:2, AR290:2, AR288:2, AR288:2, AR282:2, AR277:2, AR175:2, AR039:2, AR275:2, AR277:2, AR211:2, AR288:1, AR288:1, AR282:2, AR277:2, AR171:2, AR287:2, AR287:2, AR277:2, AR211:2, AR289:1, AR289:1, AR289:1, AR290:1, AR289:1, AR289:1, AR290:1, AR289:1, AR289:1, AR289:1, AR290:1, AR289:1, AR289:1, AR290:1, AR289:1, AR290:1, AR289:1, AR290:1, AR289:1, AR289:1, AR289:1, AR290:1, AR290:1, AR289:1, AR289:1, AR290:1, AR290:1, AR290:1, AR289:1, AR289:1, AR290:1, AR290:1, AR290:1, AR290:1, AR280:1, AR290:1, H0090:1, H0019:2, L0717:1, L0779:1, H0590:1, H0574:1, H0590:1, H0060:1, H00
262	HODFN71	1194866	272	AR282:12, AR176:8, AR162:6, AR163:5, AR170:5, AR161:5, AR266:5, AR182:5, AR181:5, AR055:5, AR228:4,

Gene No.	cDNA Clone	Contig ID:	SEQ ID	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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				AR297:2, AR178:2, AR231:2, AR27:2, AR104:2, AR288:2, AR277:2, AR288:2, AR243:2, AR236:2, AR339:2, AR296:2, AR296:2, AR296:2, AR312:2, AR173:2, AR173:2, AR053:2, AR168:2, AR313:2, AR310:3, AR210:1,
 				AR258:1, AR213:1, AR174:1, AR215:1, AR218:1, AR033:1, AR240:1, AR256:1, AR308:1, AR189:1, AR252:1, AR211:1 H0615:2 and H0624:1.
	HODFN71	834999	524	
263	HOEFV61	833079	273	AR169:6, AR039:6, AR207:6, AR096:5, AR225:4, AR264:4, AR172:4, AR235:4, AR313:4, AR183:3, AR316:3, AR252:3, AR165:3, AR166:3, AR1
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				H0421:1, H0050:1, H0090:1, L5623:1, S0053:1, L3811:1, S0037:1, S0028:1 and L0748:1.
264	HOFM033	1184465	274	AR205:90, AR212:77, AR245:75, AR274:68, AR272:67, AR216:65, AR246:62, AR252:60, AR308:59, AR213:59,
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				AR266:13, AR270:13, AR229:13, AR280:13, AR277:12, AR180:12, AR000:12, AR230:12, AR220:12, AR230:12,

Tissue Distribution Library Code:Count (see Table 4 for Library Codes)	AR176:12, AR227:11, AR294:11, AR228:10, AR283:9, AR235:9, AR182:8, AR104:7, AR055:5 H0415:1					AR192:4, AR225:3, AR217:2, AR235:2, AR172:2, AR171:2, AR183:2, AR254:2, AR168:2, AR266:2, AR170:1, AR309:1, AR193:1, AR193:1, AR180:1, AR270:1, AR175:1, AR282:1, AR165:1, AR224:1, AR277:1, AR164:1, AR300:1, AR264:1, AR039:1, AR216:1, AR291:1, AR291:1, H0415:3, S0002:2, S0212:1, H0255:1, S0358:1, H0318:1, H0045:1, H0264:1, S0144:1, H0555:1 and L0741:1.				AR263:5, AR171:4, AR213:4, AR282:4, AR205:3, AR169:3, AR235:3, AR246:3, AR162:2, AR161:2, AR180:2, AR221:2, AR176:2, AR176:2, AR245:2, AR287:2, AR183:2, AR163:2, AR311:2, AR089:1, AR309:1, AR264:1, AR104:1, AR033:1, AR191:1, AR230:1 H0415:1		AR294:16, AR169:6, AR245:6, AR192:6, AR192:6, AR195:6, AR263:5, AR169:5, AR165:4, AR165:4, AR215:4, AR203:4, AR266:4, AR172:4, AR161:4, AR212:4, AR162:4, AR222:4, AR223:4, AR213:4, AR274:4, AR261:3, AR264:3, AR272:3, AR262:3, AR262:3, AR263:3, AR261:3, AR264:3, AR272:3, AR262:3, AR262:2, AR262:1, AR
SEQ ID NO:X		525	526	527		275	529	530		276	532	277
SEQ ID: NO:X		968616	906694	902639	702186	911180	905365	892308	892291	1352378	899292	931871
cDNA Clone ID		НОЕМОЗЗ	ноғмозз	НОЕМОЗЗ	НОЕМОЗЗ	HOFMT75	HOFMT75	HOFMT75	HOFMT75	HOFNC14	HOFNC14	НОГОС73
Gene No:						265				266		267

Tissue Distribution Library Code: Count (see Table 4 for Library Codes)				AR255:8, AR263:7, AR235:7, AR170:7, AR214:7, AR163:7, AR171:7, AR245:6, AR19:6, AR19:10, AR255:6, AR225:6, AR225:6, AR2225:5, AR122:5, AR122:5, AR1225:4, AR261-5, AR261-5, AR166:5, AR225:6, AR2225:6, AR2225:5, AR180:4, AR180:4, AR180:4, AR180:4, AR20:4, AR180:4, AR20:4, AR180:4, AR20:4, AR180:4, AR20:4, AR180:4, AR20:1-4, AR20:4, AR20:4, AR089:4, AR20:4, AR089:4, AR20:4, AR089:4, AR20:4, AR089:4, AR20:4, AR089:4, AR20:4, AR089:4, AR20:4, AR20:4, AR20:4, AR20:4, AR20:4, AR089:4, AR20:4, AR2		AR214:397, AR225:322, AR215:287, AR216:269, AR223:260, AR308:259, AR311:230, AR217:200, AR222:198, AR211:192, AR210:191, AR172:189, AR166:175, AR272:173, AR212:172, AR224:160, AR274:154, AR170:151, AR264:144, AR171:143, AR168:140, AR245:137, AR242:131, AR173:129, AR221:128, AR247:127, AR169:124, AR165:119, AR218:119, AR254:116, AR188:114, AR309:113, AR181:113, AR164:112, AR213:104, AR312:103, AR196:103, AR189:103, AR178:103, AR205:99, AR191:98, AR162:97, AR190:95, AR207:94, AR269:78.
SEQ ID NO:X	533	534			536	279
SEQ Dontig ID: NO:X	907073	907072	878863	895880	902295	625973
cDNA Clone	C73	HOFOC73	HOFOC73	нодск63	HOGCK63	нонв Ү 12
Gene No:				268		269

Gene	cDNA Clone	Contig ID:	SEQ ID NO:X	
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				AR238.24, AR233.24, AR294.23, AR229.23, AR257.23, AR228.23, AR227.23, AR252.22, AR289.22, AR258.22,
			_	AKU01:17, AK280:10, AK703:3, AK263:0, AK244:3, AK104:3, AK273:3, AK201:1, AK280:10, AK703:2; AK703:2; AK701:1, AK703:2; AK701:1, AK703:2; AK701:1, AK703:2; AK701:1, AK703:2; AK701:1, AK703:2; AK703:2; AK703:2; AK701:1, AK703:2; AK701:1, AK703:2; AK701:1, AK703:2;
270	HONAH29	1299928	280	AR313:25, AR173:20, AR180:19, AR161:17, AR162:17, AR163:16, AR269:16, AR039:16, AR165:16, AR300:16,
				[AR164:15, AR225:15, AR166:15, AR182:15, AR183:15, AR179:14, AR258:14, AR223:14, AR096:14, AR270:13,
				AR257:13, AR299:13, AR238:13, AR229:13, AR1/3:13, AR262:13, AR089:13, AR191:14, AR247:12, AR206:12,
				ARI/8:12, AR293:12, AR1/8:12, AR183:12, AR242:12, AR234:12, AR297:12, AR293:10, AR193:10, AR233:10, AR231:10, AR231:10, AR231:10, AR231:10, AR231:10, AR231:10, AR331:10, AR331:
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				AR226:8, AR266:8, AR213:8, AR203:8, AR277:8, AR174:8, AR189:8, AR254:8, AR267:8, AR033:7, AR053:7,
				AR291:7, AR104:7, AR264:7, AR200:7, AR239:7, AR282:7, AR312:7, AR295:7, AR274:7, AR177:6, AR214:6,
				AR190:6, AR207:6, AR289:6, AR195:6, AR235:6, AR275:6, AR055:6, AR256:6, AR212:6, AR188:5, AR230:5,
				AR263:5, AR201:5, AR171:5, AR271:5, AR205:5, AR261:5, AR227:5, AR172:5, AR283:4, AR170:4, AK211:4,
				AR252:4, AR309:4, AR243:4, AR210:4, AR216:4, AR232:4, AR311:4, AR168:4, AR224:4, AR308:3, AR222:3,
				AR217:3, AR061:3, AR221:3, AR169:3, AR197:3, AR250:3, AR272:3, AR204:2, AR198:2, AR246:2, AR192:1
				LU/39:3, HU351:2, S3014:2, S0040:1, S0212:1, S0370:1, S0132:1, M019:1, M0413:1, M040:1, M020:1, M020:1, M020:1, M040:1, M040:1
				H0754:1, H0696:1, L0748:1, L0757:1 and H0444:1.
	HONAH29	457167	537	
271	НООВ182	1352356	281	AR207:16, AR197:15, AR309:14, AR195:13, AR311:13, AR263:13, AR24:13, AR264:13, AR245:13, AR223:12, AR23:15, AR25:17 AR25:17 AR25:17 AR25:10 AR170:10 AR171:10 AR221:10 AR053:10
				AR312:10, AR172:9, AR308:9, AR198:9, AR169:9, AR225:9, AR168:9, AR242:9, AR214:9, AR215:9, AR177:9,
				AR192:9, AR212:9, AR272:9, AR165:9, AR295:8, AR196:8, AR089:8, AR166:8, AR271:8, AR261:8, AR216:8,
				AR164:8, AR210:8, AR200:7, AR199:7, AR213:7, AR218:7, AR277:7, AR254:7, AR288:7, AR176:7, AR219:7,
				AR316:7, AR193:7, AR274:7, AR240:7, AR1881:7, AR1881:7, AR236:7, AR217:7, AR282:7, AR204:7, AR178:7,
				AR211:6, AR291:6, AR275:6, AR286:6, AR162:6, AR161:6, AR287:9, AR000:9, AR247:9, AR2703:9, AR0306:9, AR287:6, AR2805:9, AR0306:4, AR2805:9, AR2805
				AR230.6, AR103:0, AR243:0, AR103:0, AR103:0, AR103:0, AR230:0, AR230:0, AR103:0, AR1

Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
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				L0521:1, L0768:1, L0803:1, L0806:1, L0803:1, L0023:1, L0033:1, L0033:1, L0027:1, L0027:1, L0757:1, L0750:1, H0522:1, H0522:1, H0134:1, H0214:1, L0749:1, L0750:1, H0667:1, H0542:1, H0422:1, H0422:1, H0422:1, H0542:1, H0542:1, H0423:1, H0422:1, H0542:1, H05
	HOQBJ82	858338	538	
	HOQB182	857453	688	
272	HOSBY40	589431	282	AR197:6, AR309:6, AR250:5, AR176:5, AR245:4, AR169:4, AR161:4, AR162:4, AR277:4, AR163:4, AR201:4,
				AR282:4, AR233:4, AR198:4, AR17:4, AR229:3, AR212:3, AR101:3, AR082:3, AR237:3, AR238:3, AR300:3, AR269:3, AR239:3, AR299:3, AR189:3, AR246:3, AR239:3, AR239:3, AR238:3, AR300:3,
				AR313:3, AR165:3, AR270:3, AR172:3, AR166:3, AR271:3, AR275:3, AR255:3, AR240:3, AR207:2, AR274:2,
				AR216:2, AR228:2, AR312:2, AR215:2, AR183:2, AR196:2, AR226:2, AR311:2, AR096:2, AR203:2, AR262:2, AR262:2, AR101:5, AR2047:5, AR266:2, AR266:2, AR316:2, AR199:2, AR188:2, AR243:2, AR205:2, AR261:2, AR231:2,
				AR178:2, AR180:2, AR227:2, AR223:2, AR263:2, AR265:2, AR222:2, AR061:2, AR164:2, AR258:2,
				AR217:1, AR200:1, AR213:1, AR224:1, AR174:1, AR283:1, AR182:1, AR267:1, AR171:1, AR185:1, AR234:1, AR192:1 AR297:1, AR170:1 S0418:1, H0393:1, S0003:1, L0766:1, L0804:1 and S0052:1.
273	HOSDJ25	854234	283	AR207:16, AR263:14, AR235:13, AR224:13, AR225:13, AR309:12, AR196:12, AR311:12, AR214:12, AR223:12,
				AR1/2:12, AR240:11, AR100:11, AR21/1:11, AR204:11, AR1/1:11, AR102:10, AR197:10, AR161:10, AR169:10, AR222:10, AR295:10, AR288:10, AR169:10, AR204:10, AR169:10, AR169:10, AR169:10, AR204:10, AR169:10, AR169
				AR162:10, AR261:9, AR216:9, AR163:9, AR165:9, AR205:9, AR210:9, AR236:9, AR177:9, AR198:9, AR164:9,
				AR089:9, AR191:9, AR245:9, AR201:9, AR242:9, AR212:9, AR166:8, AR188:8, AR280:8, AR240:8, AR1/4:8,
				AR252:8, AR290:8, AR271:8, AR250:8, AR260:6, AR170:8, AR217:8, AR285:9, AR280:1, AR287:7, AR293:7, AR293:7,
				AR213.7, AR262.7, AR313.7, AR180.7, AR300.7, AR269.7, AR257.7, AR193.7, AR231.6, AR275.6, AR296.6,

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SEQ ID NO:X		540	284
Contig ID:		566845	614040
cDNA Clone		HOSDJ25	HOSFD58
Gene No:			274

Gene No:	cDNA Clone	Contig ID:	SEQ NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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	HOSFD58	383513	541	
275	ноисо17	429229	585	
276	HPBCU51	411080	286	
277	HPDDC77	1306899	287	AR060:25, AR104:24, AR089:24, AR055:22, AR185:18, AR039:15, AR096:12, AR316:11, AR218:9, AR283:9, AR300:9, AR219:8, AR299:8, AR240:7, AR282:7, AR207:7, AR161:6, AR162:6, AR313:6, AR163:6, AR215:6, AR198:6, AR197:6, AR204:6, AR277:5, AR201:5, AR269:5, AR228:5, AR233:5, AR236:5, AR176:4, AR309:4, AR198:6, AR171:4, AR180:4, AR182:4, AR229:4, AR257:4, AR181:4, AR237:4, AR178:4, AR255:4,

Gene No:	cDNA Clone	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
				AR183:4, AR239:4, AR275:4, AR192:4, AR287:4, AR287:4, AR177:4, AR165:4, AR268:4, AR179:4, AR270:4, AR293:3, AR166:3, AR288:3, AR222:3, AR311:3, AR191:3, AR171:3, AR266:3, AR230:3, AR213:3, AR266:3, AR213:3, AR266:3, AR293:3, AR293:2, H0659:2, H065:3, L0743:2, L066:3, L0773:1, H0633:1, H0627:1, H0629:1, H0729:1, H0729:1, H0729:1, L0659:1, L0663:1, L0773:1, L0653:1, H0644:1, H0702:1, R091:1, H0948:1, L0653:1, L0653:1, L0663:1, L0773:1, L0653:1, H0658:1, H0658:
	HPDDC77	422936	542	
278	HPEAD48	520367	288	AR196:9, AR161:9, AR162:9, AR163:8, AR173:8, AR169:8, AR171:8, AR313:7; AR168:7, AR223:7, AR175:6, AR263:6, AR260:6, AR268:6, AR264:5, AR261:5, AR262:5, AR267:5, AR269:5, AR176:5, AR166:5, AR240:6, AR282:5, AR164:5, AR214:5, AR264:5, AR261:5, AR267:5, AR267:5, AR166:5, AR274:4, AR165:5, AR300:5, AR174:4, AR181:4, AR217:4, AR191:4, AR217:4, AR191:4, AR217:4, AR287:4, AR189:4, AR218:4, AR218:3, AR206:3, AR218:3, AR206:3, AR218:3, AR206:3, AR2
279	HPEAD79	520202	289	AR277:24, AR176:5, AR039:5, AR162:5, AR205:5, AR161:5, AR225:5, AR165:5, AR582:5, AR509:5, AR168:4, AR223:4, AR228:4, AR181:4, AR266:4, AR182:4, AR269:4, AR229:4, AR257:3, AR233:3, AR272:3, AR178:3, AR180:3, AR185:3, AR264:3, AR266:3, AR

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Gene No:	Gene cDNA Clone No: ID	Contig ID:		Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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				AR200:2, AR217:2, AR246:2, AR055:2, AR175:2, AR211:2, AR232:2, AR061:2, AR283:2, AR286:2, AR053:1,
				AR222:1, AR263:1, AR256:1, AR260:1, AR258:1, AR210:1, AR291:1, AR188:1, AR174:1, AR312:1, AR252:1, AR224:1, AR219:1 H0165:1
280	HPEBE79	519003	290	AR264:5, AR290:4, AR039:4, AR268:4, AR235:4, AR299:4, AR163:4, AR096:4, AR162:4, AR282:3, AR272:3,
				AR309:3, AR253:3, AR183:3, AR213:3, AR313:3, AR300:3, AR267:3, AR225:3, AR263:2, AR221:2, AR261:2,
				AK201:2, AK245:2, AK101:2, AK005:2, AK270:2, AK247:2, AK202:2, AK251:2, AK191:2, AK200:2, AK205:2, AK201:2, AK200:2, AK2
				AR289:1, AR295:1, AR268:1, AR168:1, AR205:1, AR190:1, AR316:1, AR189:1, AR196:1, AR199:1,
		·		AR286:1, AR166:1, AR296:1, AR175:1, AR294:1 H0673:2, H0166:1 and L0517:1.
281	HPIB015	1310868	291	AR240:10, AR211:10, AR178:9, AR270:8, AR221:8, AR295:7, AR235:7, AR161:7, AR162:7, AR189:7, AR163:7,
				AR288:7, AR255:6, AR191:6, AR175:6, AR293:6, AR096:6, AR183:6, AR182:6, AR188:6, AR269:5, AR236:5,
				AR190:5, AR173:5, AR180:5, AR165:5, AR174:5, AR290:5, AR164:5, AR274:5, AR166:5, AR060:5, AR261:5,
				AR179:5, AR203:5, AR195:5, AR222:5, AR055:4, AR193:4, AR181:4, AR297:4, AR291:4, AR171:4, AR197:4,
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				AR177:4, AR299:4, AR176:4, AR033:4, AR246:4, AR172:4, AR225:3, AR263:3, AR286:3, AR275:3, AR217:3,
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				AR214:3, AR238:3, AR224:3, AR245:3, AR233:3, AR210:3, AR272:3, AR201:3, AR254:3, AR309:3, AR311:3,
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				AR200:2, AR237:2, AR231:2, AR283:2, AR229:2, AR061:2, AR239:2, AR216:2, AR227:2, AR232:2, AR226:2,
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				L0769:3, L0731:3, S0212:2, L0770:2, L0803:2, H0144:2, L0756:2, H0624:1, H0171:1, S0282:1, H0776:1, H0592:1,
				H0427:1, H0575:1, H0041:1, H0124:1, H0163:1, H0038:1, L0637:1, L0774:1, L0775:1, L0791:1, H0648:1, H0756:1,
	HPIBO15	590741	543	20026:1, L0439:1, L0777:1 and 30436:1.
282	HPJB133	68289	292	T
				ARZOW-15, ARZ/1015, ARZ/1515, ARZ/1515, ARZ/1415, ARZ/14
				AK240.3, AK312.4, AK203.4, AK234.4, AK309.4, AK103.4, AK090.4, AK177.4, AK103.4, AK102.4, AK230.4, AK304.4, AK3
				AK209.4, AK116.4, AK202.4, AK292.4, AK212.3, AK291.3, AK200.3, AK190.3, AK190.3, AK200.3, AK190.3, AK104.3, AK104.3, AK204.3, AK104.3, AK1
				[AR311.2, AR169.2, AR316.2, AR255.2, AR033.2, AR295.2, AR191.2, AR267.2, AR168.2, AR171.2,
				AR277.2, AR286.2, AR290.2, AR262.2, AR199.2, AR227.2, AR189.2, AR239.2, AR203.2, AR257.2, AR285.2,

Gene No.	cDNA Clone	SEC ID Contig ID:	SEQ ID NO.X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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283	HPJBK12	1011467	293	AR215:5, AR197:4, AR039:4, AR309:4, AR245:4, AR161:3, AR162:3, AR163:3, AR204:3, AR165:3, AR255:3, AR169:3, AR264:3, AR282:3, AR272:3, AR089:3, AR180:3, AR213:3, AR172:3, AR255:2, AR166:2, AR212:2, AR193:2, AR252:2, AR271:2, AR312:2, AR164:2, AR060:2, AR240:2, AR216:2, AR266:2, AR201:2, AR205:2, AR176:2, AR195:2, AR223:2, AR283:2, AR277:1, AR311:1, AR247:1, AR313:1, AR242:1, AR199:1, AR399:1, AR316:1, AR188:1, AR104:1, AR168:1, AR185:1, AR287:1, AR287:1, AR294:1, AR230:1, AR096:1, AR162:2
	HPJBK12	525375	544	
	HPJBK12	796925	245	
	HPJBK12	699587	246	
284	HPMDK28	846357	294	AR055.9, AR089.9, AR218:7, AR060:7, AR104:7, AR219:7, AR299:6, AR086:6, AR185:5, AR316:4, AR313:4, AR180:4, AR089:9, AR282:4, AR283:3, AR198:3, AR165:3, AR255:3, AR242:2, AR207:2, AR300:2, AR180:4, AR039:4, AR282:4, AR283:3, AR198:3, AR169:3, AR165:3, AR242:2, AR240:2, AR240:2, AR166:2, AR217:2, AR277:2, AR286:2, AR270:2, AR270:2, AR269:1, AR295:1, AR170:1, AR297:1, AR177:1, AR269:1, AR269:1, AR295:1, AR170:1, AR297:1, AR177:1, AR269:1, AR269:1, AR295:1, AR170:1, AR297:1, AR177:1, AR289:2, AR277:1, AR164:1, AR172:1, AR261:1, AR269:1, AR269:1, AR295:1, AR170:1, AR297:1, AR177:1, AR269:1, AR297:1, AR269:1, AR297:1, AR2
	HPMDK28	639118	547	
285	HPRAL78	1352342	295	AR104:11, AR089:10, AR060:9, AR283:7, AR277:7, AR039:6, AR055:6, AR316:6, AR096:6, AR219:6, AR219:5, AR299:5, AR218:5, AR313:5, AR185:5, AR240:5, AR282:3, AR204:3, AR300:2, AR312:2, AR291:2, AR251:2, AR246:2, AR052:2, AR184:2, AR202:2, AR200:2, AR238:2, AR237:2, AR298:2, AR298:2, AR298:2, AR298:2, AR292:2, AR298:2, AR298:2, AR298:1, A

Tissue Distribution Library Code:Count (see Table 4 for Library Codes)	H0051:1, H0355:1, T0006:1, H0644:1, H0032:1, H0212:1, L0456:1, H0124:1, H0708:1, S0036:1, H0038:1, H0616:1, H0087:1, H0087:1, H0089:1, L0638:1, L0637:1, L3566:1, L0087:1, H0089:1, H0089:1, L0648:1, L0638:1, L0637:1, L3566:1, L0761:1, L0772:1, L0648:1, L0803:1, L0650:1, L0809:1, L0647:1, L0665:1, H0539:1, H0521:1, H0696:1, H0553:1, L0749:1, L0749:1, L0753:1, L07			AR296:40, AR291:16, AR295:15, AR289:12, AR255:11, AR266:111, AR266:111, AR165:11, AR267:11, AR164:11, AR162:11, AR163:10, AR193:8, AR297:10, AR164:11, AR164:11, AR161:11, AR162:11, AR263:10, AR191:8, AR297:10, AR164:11, AR164:11, AR162:11, AR263:9, AR219:8, AR297:8, AR297:8, AR287:8, AR287:8, AR287:8, AR287:8, AR287:8, AR286:8, AR297:17, AR096:7, AR311:6, AR310:7, AR362:7, AR196:7, AR316:7, AR316:7, AR316:7, AR192:7, AR298:6, AR171:6, AR199:6, AR299:6, AR199:6, AR298:5, AR199:6, AR298:5, AR199:6, AR298:5, AR199:6, AR298:5, AR199:6, AR298:5, AR298:5, AR398:4, AR399:4, AR298:4, AR2		AR169:5, AR282:4, AR253:4, AR266:4, AR221:3, AR198:2, AR245:2, AR295:2, AR272:2, AR285:2, AR176:2, AR225:2, AR286:2, AR289:2, AR300:2, AR214:1, AR287:1, AR055:1, AR182:1, AR199:1, AR212:1, AR269:1, AR170:1, AR178:1, AR297:1, AR161:1, AR293:1, AR162:1:1 and L0759:1.	AR271:22, AR061:18, AR197:18, AR104:17, AR199:16, AR195:16, AR192:14, AR033:14, AR238:12, AR246:12, AR226:12, AR226:12, AR272:11, AR188:10, AR239:10, AR089:10, AR185:10, AR253:9, AR161:9, AR162:9, AR196:9, AR198:9, AR292:9, AR292:8, AR201:8, AR17:8, AR17:8, AR242:8, AR254:8, AR164:8, AR247:8, AR189:8, AR165:7, AR27:7, AR227:7, AR185:7, AR223:7, AR215:7, AR253:6, AR275:6, AR231:6, AR207:6, AR053:6,
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Contig ID:		844216	484735	829136	720095	526310	634353
cDNA Clone ID		HPRAL78	HPRAL78	HPRBC80	HPRBC80	HPRSB76	HPTVX32
Gene No:				286		287	288

SEQ Tissue Distribution Library Code:Count ID Contig ID: NO:X (See Table 4 for Library Codes)		299 AR192:5, AR242:3, AR214:3, AR195:2, AR264:2, AR168:2, AR225:2, AR277:2, AR257:1, AR172:1, AR282:1, AR171:1, AR255:1, AR275:1, AR276:1, AR296:1, AR165:1, AR182:1, AR224:1, AR295:1, AR295:1	300 AR104:20, AR272:17, AR185:15, AR293:14, AR237:14, AR230:13, AR296:13, AR161:12, AR234:12, AR162:12, AR283:12, AR163:12, AR294:12, AR294:12, AR294:11, AR228:11, AR233:10, AR297:10, AR096:10, AR252:10, AR289:9, AR061:9, AR231:9, AR239:9, AR165:9, AR308:9, AR164:9, AR257:9, AR237:9, AR166:8, AR275:8, AR235:8, AR313:8, AR060:8, AR055:8, AR291:8, AR169:7, AR089:7, AR177:7, AR311:7, AR265:7, AR254:7, AR287:7, AR262:7, AR285:7, AR285:5, AR312:5, AR300:6, AR316:6, AR296:5, AR296:5, AR285:5, AR285:5, AR285:5, AR285:5, AR285:5, AR285:5, AR285:5, AR285:5, AR286:5, AR296:5, AR296:6, AR296:5, AR296:5, AR196:3, AR196:3, AR296:3, AR196:3, AR196:3, AR196:3, AR196:3, AR196:3, AR296:3, AR196:3, AR296:1, AR296:	551		552
		526749	1001560	876469	789574	413270
cDNA Clone		HPVAB94	HPWAY46	HPWAY46	HPWAY46	1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1
Gene No:	1					

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Gene No:	cDNA Clone ID	Contig ID:	_	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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				AR263:4, AR262:4, AR205:4, AR275:4, AR196:4, AR174:4, AR237:4, AR179:4, AR312:4, AR189:4, AR214:4,
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				AR226:3, AR286:3, AR207:3, AR039:3, AR195:3, AR055:3, AR197:3, AR252:3, AR218:3, AR277:3, AR225:3,
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292	HPWDJ42	722246	302	AR313:65, AR165:39, AR164:38, AR166:36, AR162:32, AR161:32, AR163:31, AR096:29, AR089:29, AR242:27,
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				AR179:18, AR299:18, AR258:17, AR193:17, AR185:17, AR182:17, AR238:17, AR234:17, AR312:16, AR183:16,
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				AR274:13, AR233:12, AR269:12, AR297:12, AR268:12, AR282:11, AR260:11, AR230:11, AR285:11, AR296:11,
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	HPWDJ42	692213	554	
293	HPZAB47	585702	303	AR313:12, AR165:9, AR164:8, AR163:8, AR166:8, AR162:8, AR173:8, AR161:7, AR242:7, AR089:7, AR180:6,
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Gene No:	cDNA Clone	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
				AR055:1, AR230:1, AR225:1, AR211:1 L0530:2, S0470:1, S0360:1, 10003:1, H0488:1, L0789:1, S0578:1 and S0168:1.
294	HRAAB15	658717	304	AR184:5, AR263:5, AR170:5, AR171:4, AR311:4, AR265:4, AR165:4, AR221:4, AR164:4, AR166:4, AR243:4, AR308:4, AR308:4, AR325:3, AR252:3, AR162:3, AR169:3, AR161:3, AR195:3, AR163:3, AR269:3,
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				AR257:2, AR247:2, AR060:2, AR231:1, AR173:1, AR226:1, AR191:1, AR298:1, AR288:1, AR190:1, AR039:1,
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				H0580:1, S0222:1, H0551:1, L0770:1, L0796:1, L0800:1, L0804:1, L0655:1, H0555:1 and L0779:1.
295	HRABA80	882176	305	i –
				AR316:387, AR039:363, AR240:317, AR277:285, AR300:278, AR218:153, AR313:152, AR219:140, AR242:4,
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	HRABA80	588460	555	- 1
296	HRACD15	871221	306	_
) 			· ·	
				AR291:7, AR212:7, AR297:7, AR264:7, AR288:7, AR199:7, AR197:7, AR282:7, AR300:6, AR240:6, AR272:6,
				AR196:6, AR285:6, AR275:6, AR201:6, AR200:6, AR205:0, AR225:0, AR225:0, AR225:0, AR225:0, AR165:0, AR265:0, AR2
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				AR250:3, AR233:3, AR216:3, AR204:2, AR226:2, AR221:2, AR232:2, AR00:1:4, AR226:2, AR211:2, AR111:2, AR2104:2, AR210:3, AR210:3, AR210:3, AR200:3, A
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Gene No:	cDNA Clone ID	Contig ID:	SEQ ID NO:X	
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				H0087:1, H0100:1, H0429:1, S0016:1, H0561:1, H0132:1, H0646:1, S0422:1, L0598:1, H0529:1, L0763:1, L0638:1, L4747:1, L0761:1, L0800:1, L0648:1, L0774:1, L0651:1, L0378:1, L0776:1, L0761:1, L0800:1, L0648:1, L0774:1, L0651:1, L0378:1, L0776:1, L0791:1,
			_	L0663:1, H0144:1, H0593:1, H0689:1, H0659:1, S0406:1, S0037:1, L0745:1, L0779:1, L0752:1, L0755:1, S0394:1, L0593:1, S0026:1, H0665:1, H0542:1, H0542:1 and H0506:1.
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700	LID A CT25	99776	307	AR1723-51 AR274-51 AR271-38 AR273-34 AR275-30 AR172:14 AR171:9 AR170:9 AR182:9 AR215:9.
167	CCCANII	000//0	<u>}</u>	
				AR266.5, AR175.4, AR270.4, AR165.4, AR164.4, AR181.4, AR166.4, AR290.4, AR163.4, AR238.4, AR096.4,
			•	AR161:4, AR162:4, AR195:3, AR267:3, AR274:3, AR291:3, AR243:3, AR250:3, AR289:3, AR179:3, AR316:3,
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				AR227:1, AR201:1, AR312:1, AR200:1, AR039:1, AR188:1, AR239:1, AR296:1, AR193:1 L0731:11, L0803:7,
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	HRACJ35	730504	557	
	HRACJ35	470546	558	
298	HRGBL78	910133	308	AR052:15, AR213:14, AR053:10, AR244:8, AR096:7, AR184:6, AR215:6, AR310:5, AR251:5, AR241:5, AR221:4,

HRGBL78 904040 559 HRGBL78 904621 560 HRGBL78 863802 561 299 HROAJ39 1181699 309		X CX	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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	904621	560	
	863802	561	
	1181699		AR055:8, AR060:6, AR218:6, AR300:5, AR316:4, AR089:4, AR240:4, AR282:3, AR185:3, AR104:3, AR299:3, AR313:3, AR096:3, AR283:3, AR039:2, AR219:2, AR277:2 H0316:1, L3905:1, L0565:1, L0438:1, H0521:1, L0439:1 and L0594:1.
HROAJ39	1114849	562	
HROAJ39	1027712	563	
300 HROBD68	827306	310	AR196:23, AR161:12, AR162:12, AR163:11, AR242:9, AR165:8, AR164:8, AR166:8, AR191:8, AR089:8, AR275:8, AR096:7, AR181:7, AR175:7, AR089:6, AR173:6, AR264:6, AR060:6, AR258:5, AR089:8, AR275:8, AR096:7, AR181:7, AR175:7, AR089:6, AR180:5, AR264:6, AR060:6, AR258:5, AR174:5, AR236:5, AR276:5, AR198:5, AR277:5, AR198:5, AR270:5, AR270:5, AR270:5, AR270:5, AR270:5, AR270:5, AR270:5, AR280:5, AR280:5, AR280:5, AR280:4, AR280:4, AR280:4, AR280:4, AR280:4, AR280:4, AR280:4, AR280:4, AR260:4, AR260:4, AR260:4, AR280:4, AR280:4, AR280:3, AR271:4, AR271:4, AR271:4, AR291:3, AR271:3, AR290:3, AR280:3,

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305	HSDAJ46	692358	315	AR162:9, AR161:9, AR163:8, AR165:7, AR215:7, AR164:7, AR166:7, AR261:7, AR288:7, AR221:7, AR255:6, AR297:6, AR180:6, AR176:6, AR089:5, AR216:5, AR181:5, AR184:5, AR196:5, AR214:5, AR039:5, AR235:5, AR066:5, AR295:5, AR295:5, AR297:4, AR257:4, AR313:4, AR287:4, AR287:4, AR257:4, AR313:4,
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306	HSDEK49	1352253	316	AR290:45, AR268:37, AR240:23, AR267:22, AR269:16, AR270:14, AR234:10, AR055:10, AR238:10, AR184:9, AP300:8, AP170:8, AP170:3, AP180:3, AP170:4, AP180:5, AP170:4, AP180:5, AP170:4, AP180:5, AP170:5, AP1
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				\$0426:2, L0775:2, \$0330:2, L0747:2, L0779:2, \$0260:2, L0599:2, L0603:2, H0739:1, H0170:1, \$0110:1, \$0334:1, \$03444.1 13444.1 13444.1 H030:1, H0644.1 H0644.1 H0644.1
				H0090:1, H0063:1, S0142:1, L0770:1, L0769:1, L0651:1, L0776:1, L0659:1, L0519:1, L0664:1, H0682:1, L0749:1, L0752:1. S0031:1 and H0506:1.
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307	HSDEZ20	1352287	317	AR176:5, AR252:5, AR266:5, AR215:4, AR223:4, AR181:4, AR197:4, AR161:4, AR162:4, AR264:4, AR163:3, AR235:3, AR165:3, AR166:3, AR309:3, AR207:3, AR267:3, AR214:3, AR228:3, AR182:3, AR254:3,

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Contig ID:		704101	834619	836071	589974	795252
cDNA Clone		HSDEZ20	HSDFJ26	HSDFJ26	HSDFW45	HSDJA15
Gene No:	·		308		309	310

Contig ID: NO:X ID
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313	HSDSE75	545057	323	AR096:3, AR225:3, AR266:3, AR055:3, AR060:3, AR309:2, AR170:2, AR222:2, AR104:2, AR214:2, AR254:2, AR163:2, AR161:2, AR195:2, AR282:2, AR089:1, AR224:1, AR283:1, AR275:1, AR228:1, AR240:1, AR290:1, AR195:1, AR165:1, AR166:1, AR216:1, AR2
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314	HSFAM31	552789	324	AR173:8, AR178:6, AR183:6, AR313:6, AR293:6, AR299:6, AR180:6, AR182:5, AR270:5, AR175:5, AR269:5, AR162:5, AR161:5, AR181:5, AR163:5, AR291:4, AR282:4, AR176:4, AR238:4, AR165:4, AR26:4,
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				AR224.2, AR060:2, AR190:2, AR232.2, AR216:2, AR207:2, AR168:2, AR172:1, AR311:1, AR055:1, AR256:1, AR234:1, AR197:1, AR197:1, AR205:1, AR205:1, AR194:1, H0154:1, and H0087:1.
315	HSICV24	1352248	325	AR253:24, AR252:13, AR254:13, AR250:9, AR266:6, AR221:6, AR308:5, AR212:5, AR263:5, AR245:4, AR053:4,
				AR312:4, AR271:4, AR180:4, AR270:3, AR205:3, AR171:3, AR272:3, AR193:3, AR225:3, AR264:3, AR290:2,
				AR316:2, AR224:2, AR179:2, AR216:2, AR313:2, AR161:2, AR177:2, AR163:1, AR299:1, AR165:1, AR277:1,
				AR182:1, AR296:1, AR291:1, AR237:1, AR175:1, AR219:1, AR261:1, AR201:1, AR039:1, AR257:1, AR164:1 H0036:1
	HSICV24	612877	571	
316	HSIDJ81	589447	326	AR313:41, AR039:35, AR096:26, AR173:25, AR299:21, AR258:20, AR180:20, AR185:19, AR089:18, AR262:18, AR161:18, AR162:18, AR162:18, AR179:18, AR269:17, AR360:17, AR360:
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				AR282:9, AR230:9, AR275:9, AR290:8, AR264:8, AR251:8, AR251:8, AR237:8, AR242:9, AR190:8, AR192:9, AR288:7, AR288:7, AR235:7, AR295:7, AR291:7, AR228:7, AR235:7, AR033:7, AR295:6, AR227:6,
				AR263:6, AR266:6, AR197:5, AR211:5, AR308:5, AR053:5, AR256:5, AR250:5, AR232:4, AR210:4, AR272:4,
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	DNA Clone		SEQ	Tissue Distribution Library Code: Count
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317	HSKCP69	702021	327	AR178:6, AR180:9, AR170:6, AR270:6, AR174:6, AR180:1, AR180:1, AR180:1, AR180:1, AR180:1, AR180:1, AR180:1, AR180:1, AR180:1, AR270:1, AR280:1, AR2
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	HSKCP69	413210	572	
318		1352409	328	AR039:106, AR104:103, AR055:103, AR240:102, AR060:87, AR096:84, AR282:77, AR283:67, AR300:66, AR316:57, AR185:48, AR219:45, AR218:44, AR089:40, AR299:36, AR277:34, AR313:31 S0212:13, S0126:12, L0777:11, S0027:10, S0028:10, S0250:7, H0717:6, L0662:6, L0747:6, S0360:5, S0022:5, S0206:5, L0779:5, S0194:5, L0659:4, L0751:4, L0731:4, L0758:4, H0713:3, H0716:3, S0444:3, H0599:3, L0163:3, S0210:3, L0807:3, S0390:3, S0037:3, S3014:3, L0740:3, S0192:3, H0295:2, H0786:2, H0706:2, H0706:2, H0309:2, H0023:2, H0373:2, H0266:2, H0039:2,

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				L0438:1, H0519:1, S0152:1, S0454:1, H0521:1, H0696:1, S3012:1, S0124:1, L0439:1, L0750:1, H0595:1, S0436:1, H0668:1, H0667:1, S0242:1, S0276:1 and L3603:1.
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	HSKDA27	872570	574	
319	HSKGN81	676075	329	AR252:303, AR263:240, AR211:227, AR272:220, AR210:215, AR216:184, AR253:180, AR250:170, AR264:169,
}				AR242:163, AR172:160, AR245:160, AR274:155, AR254:148, AR247:147, AR313:142, AR165:141, AR053:139,
				AR225:136, AR195:131, AR215:129, AR221:129, AR308:124, AR197:123, AR214:123, AR212:122, AR170:119,
				AR166:118, AR224:118, AR213:117, AR171:115, AR205:113, AR312:113, AR162:109, AR217:108, AR309:106,
				AR199:106, AR271:105, AR164:100, AR198:93, AR168:92, AR169:92, AR188:92, AR207:91, AR273:91, AR173:91,
				AR256:90, AR291:89, AR163:80, AR163:86, AR246:82, AR246:82, AR511:10, AR291:10, AR251:10, AR291:10, AR291:
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				AR177:32, AR239:30, AR176:30, AR237:29, AR234:27, AR104:27, AR226:26, AR294:25, AR060:24, AR055:18,
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				S0116:1, H0341:1, H0254:1, H0255:1, H0306:1, H0402:1, S0360:1, S0408:1, S0046:1, S0132:1, H0619:1, H0549:1,
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320	HSKHZ81	1307105	330	AR218:51, AR219:48, AR210:39, AR197:35, AR275:35, AR195:29, AR211:27, AR177:24, AR198:22, AR089:21, AR175:19, AR096:19, AR191:19, AR282:19, AR192:18, AR209:18, AR096:19, AR191:19, AR282:19, AR192:18, AR209:12, AR192:14, AR292:13, AR192:13, AR292:13, AR192:11, AR292:12, AR292:12, AR292:13, AR292:13, AR292:13, AR292:11, AR292:13, AR292:11, AR292:13, AR292:11, AR292:13, AR292:11, AR292:
	HSKHZ81	552233		
321	HSLJG37	1016920	331	AR282:7, AR207:5, AR309:5, AR204:5, AR224:4, AR161:3, AR162:3, AR163:3, AR217:3, AR246:3, AR257:3, AR201:3, AR275:3, AR272:3, AR272:2, AR272:1, AR2
	HSLJG37	852244	577	
	HSLJG37	895206	578	

Tissue Distribution Library Code:Count (see Table 4 for Library Codes)	AR170:5, AR169:4, AR180:4, AR313:4, AR221:3, AR178:3, AR223:3, AR245:3, AR192:3, AR235:2, AR204:2, AR182:2, AR299:2, AR216:2, AR291:2, AR274:2, AR171:2, AR214:2, AR217:2, AR193:2, AR266:1, AR308:1, AR293:1, AR257:1, AR247:1, AR232:1, AR225:1, AR283:1, AR210:1, AR282:1 H0163:2	AR242:8, AR205:6, AR238:6, AR170:6, AR207:5, AR201:4, AR215:3, AR204:3, AR096:3, AR296:3, AR172:2, AR233:2, AR089:2, AR182:2, AR055:2, AR257:2, AR299:1, AR104:1, AR272:1, AR210:1, AR185:1, AR297:1 H0163:1		AR261:16, AR296:15, AR309:15, AR161:14, AR163:14, AR162:14, AR291:12, AR295:10, AR177:10, AR264:9,	AR287:9, AR165:9, AR285:9, AR166:9, AR297:8, AR275:8, AR164:8, AR181:8, AR288:8, AR235:8, AR196:7,	AR176:7, AR293:7, AR033:7, AR089:7, AR255:7, AR286:7, AR257:1, AR229:1, AR262:1, AR173:6, AR231:6,	AK200:0, AK312:0, AK176:0, AK233:0, AK233:0, AK200:0, AK207:0, AK197:0, AK189:3, AK239:0, AK237:5, AK271:5, AK278:6, AK240:6, AK2	AR191:5, AR226:5, AR272:5, AR174:5, AR225:5, AR274:5, AR185:5, AR268:5, AR061:5, AR179:5, AR290:5,	AR215:5, AR263:5, AR060:4, AR199:4, AR212:4, AR183:4, AR300:4, AR168:4, AR193:4, AR188:4, AR175:4,	AR246:4, AR243:4, AR299:4, AR313:4, AR203:4, AR230:4, AR055:4, AR311:4, AR282:4, AR234:4, AR28:4,	AR195:4, AR218:4, AR180:4, AR283:4, AR169:4, AR254:4, AR104:4, AR23::4, AR267:4, AR210:3,	AR213:3, AR253:3, AR182:3, AR227:3, AR245:3, AR236:3, AR039:3, AR210:3, AR256:3, AR260:3, AR170:3,	ARZ/0:3, ARZ04:3, ARZ11:3, AR1/1:3, AR2//:2, ARZ1/:2, ARU33:4, ARZ05:2, ARZ10:2, ARZ22:2, ARZ24:2 1 0347:9 1 0450:7 1 0376:4 1 0720:4 1 0460:4 1 0775:4 1 0750:4 1 0603:4 10656:3 50410:3 1 0764:3	LOFFICE LUCUST, LOT 1015 LOT 1	L000313, L043373, L073013, 3033012, 30406.2, L0471.2, M0271.2, 30440.2, L0702.2, L0702.2, L0372.2, L06341.2, L06372.2, L06472.2, L06472.	S0404:2, L0754:2, L0749:2, L0777:2, L0758:2, L0596:2, H0657:1, S0001:1, H0484:1, H0638:1, S0418:1, S0444:1,	L0717:1, H0333:1, H0156:1, H0052:1, H0545:1, H0012:1, H0083:1, H0687:1, H0674:1, H0090:1, H0063:1, H0264:1,	H0100:1, L0434:1, L0351:1, H0494:1, H0561:1, S0466:1, H0641:1, H0529:1, L0763:1, L0761:1, L0667:1, L0363:1,	L0650:1, L0653:1, L0654:1, L0379:1, L0607:1, L0807:1, L0635:1, L0783:1, L0383:1, L0809:1, L0666:1, H0658:1,	H0670:1, H0648:1, H0521:1, S0406:1, L0748:1, L0731:1, L0593:1, L0595:1, S0026:1, S0276:1 and H0422:1.	AR283:139, AR096:16, AR089:15, AR218:14, AR219:12, AR055:11, AR313:10, AR316:10, AR039:9, AR060:9,	AR104:9, AR277:8, AR240:8, AR299:8, AR382:8, AR300:6, AR185:5, AR225:5, AR225:4, AR223:4, AR215:4,	AR235:3, AR192:3, AR171:2, AR165:2, AR217:2, AR257:2, AR263:2, AR195:2, AR297:2, AR314:2, AR309:1,	AR213:1, AR267:1, AR164:1, AR166:1, AR182:1, AR269:1, AR264:1, AR173:1, AR261:1, AR291:1 L0777:3,	S0026:2, S0040:1, S0418:1, L3388:1, H0437:1, H0013:1, H0274:1, H0083:1, S0250:1, H0644:1, H0163:1, L0643:1,	LO/83:1, L3661:1, S0126:1, H0659:1, LU//9:1 and S0196:1.
SEQ ID NO:X	332 A A A	333 A A H	579	1	<u> </u>	<u> </u>	<u>६</u> ∢	≥	_∢_	<u>∢</u>	<u>∢</u> _	<u> </u>	∢	-L-	<u>-l</u> -	<u>. 02</u>	<u>l H</u>	<u>111</u>		H	335	۷.	<u> </u>	<u> </u>	<u>o</u> .	4
Contig ID:	467397	1352201	545060	H									-								1306702					
cDNA Clone Contig ID: NO:X	HSNAD72	HSNMC45	HSNMC45	HSQCM10	,														-		HSQEO84					
Gene No:		323		324																	325					

Tissue Distribution Library Code:Count (see Table 4 for Library Codes)			AR197:9, AR271:8, AR176:7, AR162:7, AR161:7, AR201:7, AR163:7, AR192:6, AR204:6, AR207:6, AR266:6, AR267:6 AR267:6, AR228:6, AR229:6, AR169:6, AR177:6, AR237:6, AR198:6, AR233:5, AR245:5, AR181:5, AR193:5,	AR250:5, AR243:5, AR053:5, AR269:5, AR239:5, AR309:5, AR089:5, AR180:5, AR264:5, AR165:5, AR214:5,	AR182:4, AR060:4, AR224:4, AR061:4, AR268:4, AR261:4, AR178:4, AR166:4, AR230:4, AR257:4, AR226:4,	AR183:4, AR164:4, AR270:4, AR275:4, AR231:4, AR236:4, AR096:4, AR179:4, AR246:4, AR289:4, AR039:4,	AR055:4, AR293:4, AR196:4, AR175:4, AR316:4, AR272:4, AR234:4, AR168:4, AR225:4, AR286:4, AR247:4,	AR312:4, AR212:4, AR255:4, AR296:4, AR242:4, AR294:3, AR300:3, AR290:3, AR185:3, AR205:3, AR291:3,	AR238:3, AR262:3, AR227:3, AR295:3, AR287:3, AR288:3, AR174:3, AR297:3, AR216:3, AR311:3, AR277:3,	AR170:3, AR191:3, AR285:3, AR188:3, AR213:3, AR215:3, AR313:3, AR217:3, AR308:3, AR232:3, AR203:3,	AR195:3, AR282:3, AR173:2, AR033:2, AR172:2, AR189:2, AR171:2, AR274:2, AR223:2, AR190:2, AR299:2,	AR104:2, AR211:2, AR258:2, AR200:2, AR283:2, AR263:2, AR256:2, AR221:2, AR199:2, AR240:2, AR222:2,	AR210:2, AR253:1, AR254:1, AR260:1, AR219:1, AR218:1 S0007:1, H0555:1 and S0026:1.	AR225:4, AR309:4, AR060:4, AR192:3, AR235:3, AR162:3, AR055:3, AR161:3, AR163:3, AR215:3, AR275:3,	AR169:3, AR254:3, AR217:3, AR233:2, AR170:2, AR177:2, AR181:2, AR236:2, AR255:2, AR228:2, AR180:2,	AR289:2, AR237:2, AR243:2, AR239:2, AR166:2, AR285:2, AR266:2, AR272:2, AR287:2, AR222:2, AR274:2,	AR176:2, AR061:2, AR271:2, AR223:2, AR247:2, AR214:1, AR224:1, AR240:1, AR172:1, AR213:1, AR283:1,	AR262:1, AR295:1, AR033:1, AR089:1, AR174:1, AR229:1, AR216:1, AR234:1, AR238:1, AR231:1, AR316:1,	AR218:1, AR300:1, AR293:1 S0022:4	AR225:17, AR223:16, AR215:16, AR214:14, AR224:13, AR170:13, AR217:12, AR168:12, AR172:12, AR221:12,	AR246:11, AR222:11, AR216:11, AR269:11, AR169:11, AR171:10, AR183:9, AR268:9, AR165:8, AR290:8,	AR161:8, AR164:8, AR162:8, AR270:8, AR163:8, AR166:8, AR291:7, AR244:7, AR298:7, AR282:7, AR182:7,	AR180:7, AR266:7, AR176:7, AR186:7, AR173:7, AR052:6, AR231:6, AR271:6, AR207:6, AR292:6, AR250:6,	AR228:6, AR282:6, AR238:6, AR206:6, AR061:6, AR273:6, AR296:6, AR275:6, AR243:6, AR181:6, AR247:5,	AR289:5, AR285:5, AR200:5, AR240:5, AR210:5, AR053:5, AR249:5, AR314:5, AR241:5, AR202:5, AR218:5,	AR219:5, AR235:5, AR194:5, AR178:5, AR197:5, AR089:5, AR189:5, AR177:5, AR211:5, AR239:5, AR175:5,	AR237:5, AR198:5, AR293:5, AR201:5, AR190:5, AR188:5, AR295:5, AR251:5, AR255:5, AR245:4, AR280:4,	AR254:4, AR185:4, AR196:4, AR060:4, AR272:4, AR315:4, AR313:4, AR312:4, AR300:4, AR193:4, AR309:4,	AR316:4, AR257:4, AR179:4, AR232:4, AR311:4, AR234:4, AR233:4, AR236:4, AR264:4, AR286:4, AR299:4,	AR294:4, AR204:4, AR033:4, AR229:4, AR039:4, AR226:4, AR191:4, AR205:4, AR184:3, AR288:3, AR274:3,	AR096:3, AR261:3, AR287:3, AR203:3, AR297:3, AR284:3, AR174:3, AR212:3, AR277:3, AR055:3, AR313:3,
SEQ ID NO:X	280	581		(≼	_<	_₹	<u> </u>	⋖	¥	_₹	V	_∢	_₹	337 A		_₹	_₹	_≰	_≪	338 A	⋖	_≼	_₹	_∢	_<	_≼	_∢	⋖	▼	<u>∢</u>	<u>∢</u>
Contig ID:	602258	401251	460537								t.			892171						1352343									-		
cDNA Clone Contig ID: ID ID ID	HSQE084	HSQEO84	нЅQFР66											HSRFZ57						HSSGD52									-		
Gene No:			326											327	<u> </u>					328											

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329	HSUBW09	413246	339	AR186:66, AR202:60, AR259:59, AR206:59, AR292:58, AR061:56, AR052:56, AR283:51, AR227:49, AR251:49,
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SEQ Tissue Distribution Library Code: Count (See Table 4 for Library Codes)	AR290:2, AR190:2, AR285:2, AR226:2, AR055:2, AR227:2, AR308:2, AR183:2, AR283:2, AR233:2, AR223:2, AR213:2, AR214:2, AR217:2, AR234:1, AR185:1, AR185:1, AR299:1, AR294:1, AR247:1, AR174:1, AR286:1, AR169:1,	AR196:1, AR262:1, AR203:1, AR240:1, AR168:1, AR309:1, AR189:1, AR257:1 H0309:1, H0012:1, H0038:1, L0775:1, L0793:1 and L0758:1.	341	AR272:3, AR039:3, AR192:3, AR213:3, AR253:3, AR166:3, AR264:3, AR089:3, AR282:3, AR204:3, AR235:3, AR235:3, AR235:3, AR245:3, AR255:3, AR2	AR277:2, AR104:2, AR286:2, AR246:2, AR287:2, AR033:2, AR033:2, AR243:2, AR237:2, AR233:2, AR233:2,	AR268:2, AR293:2, AR180:2, AR060:2, AR175:2, AR198:2, AR229:2, AR177:2, AR270:2, AR233:2, AR183:2,	AR228:2, AR261:2, AR239:2, AR316:2, AR285:2, AR179:2, AR232:1, AR312:1, AR061:1, AR288:1,	AR257:1, AR096:1, AR291:1, AR225:1, AR226:1, AR294:1, AR295:1, AR185:1, AR311:1, AR227:1, AR234:1, AR194:1, AR104:1, AR203:1, AR297:1, AR191:1, AR247:1, AR308:1, AR288:1, AR216:1, AR255:1, AR191:1	H0309:1	342	AR269:5, AR271:5, AR165:5, AR239:5, AR226:5, AR180:5, AR164:5, AR181:5, AR250:5, AR207:5, AR174:5,	AR299:5, AR224:5, AR246:5, AR166:5, AR266:5, AR229:5, AR104:5, AR182:4, AR189:4, AR183:4, AR238:4,	AR233:4, AR283:4, AR237:4, AR313:4, AR193:4, AR275:4, AR270:4, AR243:4, AR053:4, AR268:4, AR272:4,	AR228:4, AR300:4, AR185:4, AR282:3, AR201:3, AR267:3, AR316:3, AR191:3, AR178:3, AR190:3, AR232:3,	AR240:3, AR264:3, AR179:3, AR205:3, AR096:3, AR311:3, AR291:3, AR235:3, AR033:3, AR198:3,	AR197:3, AR227:3, AR261:3, AR1061:3, AR175:3, AR312:3, AR236:3, AR214:2, AR257:2, AR215:2, AR254:2, AR	AR28912, AR2U412, AR211112, AR24112, AR22512, AR211112, AR03912, AR21212, AR20512, AR20512, AR19513, AR195112, AR19612, AR28812, AR21812, AR28612, AR27412, AR30812, AR20612, AR21212, AR26212, AR29312, AR17112,	AR295:2, AR223:2, AR296:2, AR230:1, AR170:1, AR172:1, AR195:1, AR213:1, AR200:1, AR173:1	H0032:3, L0438:3, L0758:3, S0376:2, L0439:2, S0418:1, S0410:1, H0574:1, H0156:1, H0036:1, S0010:1, S0474:1,	H0581:1, T0110:1, S0214:1, S0036:1, H0591:1, H0634:1, H0634:1, H0494:1, L0796:1, L0372:1, L0803:1, L0804:1,	-	7 343 AR216:52, AR214:45, AR203:44, AR215:39, AR199:38, AR214:34, AR222:53, AR217:53, AR223:53, AR117:53, AR170:31 AR118:30 AR223:29 AR224:57 AR170:47 AR110:27 AR210:25 AR221:24 AR272:24,	AR175:23, AR247:22, AR246:22, AR195:21, AR213:21, AR218:20, AR212:20, AR189:19, AR211:19, A	AR175:23, AR247:22, AR246:22, AR195:21, AR213:21, AR218:20, AR212:20, AR189:19, AR211:19, AR053:19,
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	HTADX17	457172	585	
336	HTAEE28	1018291	346	AR170:5, AR169:4, AR221:3, AR250:3, AR217:3, AR242:2, AR263:2, AR171:2, AR193:2, AR245:2, AR201:2, AR172:2, AR183:2, AR300:2, AR267:1, AR367:1, AR269:1, AR264:1, AR168:1, AR161:1, AR215:1, AR311:1, H0250:3, H0069:2, L0771:2, S0404:2, H0650:1, H0656:1, H0486:1, H0013:1, H0318:1, S0422:1,
·				L0644:1, L0768:1, L0794:1, L0804:1, L0655:1, L0789:1, L0664:1, H0436:1 and L0758:1.
	HTAEE28	882919	985	
	HTAEE28	864120	287	
337	HTECC05	1352365	347	AR176:5, AR169:3, AR224:3, AR180:3, AR291:3, AR225:3, AR238:3, AR267:3, AR261:2, AR245:2, AR289:2,
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				S0049:3, H0708:3, L0773:3, L0805:3, L0809:3, L0519:3, H0670:3, L0748:3, L0731:3, L0757:3, L0581:3, H0295:2,
				H0341:2, S0444:2, S0222:2, L0622:2, H0253:2, H0309:2, T0115:2, H0544:2, H0545:2, H0081:2, H0012:2, H0673:2,
				S0036:2, H0616:2, L0770:2, L0774:2, L0518:2, H0725:2, S0374:2, H0696:2, L0588:2, H0543:2, L0615:1, H0160:1,
				H0225:1, H0713:1, S6024:1, S0430:1, H0656:1, S0116:1, S0212:1, H0483:1, H0306:1, H0638:1, H0125:1, S0420:1,
				203381, 304081, F00371, 304781, F00401, F04111, 302781, F04411, F04411, F04011, F02711, F02381, F032311, F032311, F03881, F038
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				L3904:1, L0761:1, L0772:1, L0764:1, L0767:1, L0768:1, L0766:1, L0649:1, L0803:1, L0375:1, L0806:1, L0776:1,
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Tissue Distribution Library Code:Count (see Table 4 for Library Codes)			AR245:5, AR311:4, AR282:3, AR253:3, AR089:3, AR176:3, AR272:2, AR162:2, AR164:2, AR165:2, AR299:2, AR168:2, AR271:2, AR271:2, AR166:2, AR193:2, AR204:2, AR171:2, AR266:1, AR266:1, AR297:1, AR269:1, AR216:1, AR172:1, AR224:1, AR033:1, AR163:1, AR296:1, AR178:1, AR161:1, AR283:1, AR174:1, AR181:1 L0758:2 and H0038:1.		AR174:12, AR191:12, AR190:11, AR244:11, AR181:11, AR291:10, AR186:10, AR180:10, AR175:10, AR192:10, AR189:9, AR216:9, AR240:9, AR240:9, AR241:9, AR178:9, AR270:9, AR177:8, AR266:8, AR268:8, AR288:8, AR183:8, AR273:8, AR274:8, AR165:1, AR164:7, AR164:7, AR164:7, AR165:7, AR202:7, AR202:7, AR161:7, AR163:7, AR246:7, AR246:7, AR245:6, AR187:6, AR289:6, AR187:6, AR289:6, AR187:6, AR289:6, AR289:6, AR289:6, AR289:6, AR289:6, AR289:6, AR289:6, AR289:5, AR289:5, AR289:5, AR289:6, AR289:6, AR289:6, AR196:4, AR285:5, AR289:5, AR289:5, AR289:5, AR289:5, AR289:5, AR289:5, AR289:6, AR196:4, AR285:4, AR285:4, AR286:4, AR285:4, AR286:4, AR288:4, AR287:4, AR287:4, AR288:4, AR288:3, AR287:3, AR288:3, AR287:3, AR288:3, AR287:3, AR288:3, AR	AR240:15, AR055:12, AR060:7, AR039:6, AR299:6, AR219:6, AR277:5, AR089:5, AR218:5, AR300:5, AR185:5, AR104:5, AR283:4, AR283:4, AR316:4, AR096:3, AR313:3 H0486:3, H0253:1, H0544:1, H0012:1, S0388:1, H0553:1, H0090:1, H0038:1, H0652:1, L0769:1, L0641:1, L0806:1, H0696:1, L0748:1, L0749:1, S0031:1 and S0196:1.	AR165:8, AR164:8, AR166:7, AR207:7, AR222:6, AR195:6, AR263:6, AR216:6, AR053:6, AR168:6, AR245:5, AR162:5, AR169:5, AR170:5, AR089:5, AR213:4, AR261:4, AR312:4, AR282:4, AR308:4, AR212:4, AR235:4, AR177:4, AR205:4, AR172:4, AR223:4, AR242:4, AR192:3, AR192:3, AR256:3, AR256:3, AR236:3, AR286:3, AR270:3, AR181:3, AR264:3, AR271:3, AR291:3, AR193:3, AR198:3, AR316:3, AR295:3, AR295:3, AR311:3, AR197:3, AR309:3, AR198:3, AR196:3, AR243:2, AR277:2,
SEQ ID NO:X	588		348	590		350	351
Contig ID:		666743	1352193	519372	206980	543396	908143
ં	HTECC05	\vdash	HTEDY42	HTEDY42	HTEBB42	HTEFU65	HTEGI42
Gene No:			338		339	340	341

Gene No:	cDNA Clone Contig ID:	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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	HTEGI42	904624	591	
	HTEGI42	850770	592	
	HTEGI42	847564	293	
	HTEGI42	830165		
342	нтениз1	600394	· · · · · · · · · · · · · · · · · · ·	AR052:91, AR249:84, AR251:80, AR186:76, AR259:66, AR055:65, AR248:65, AR218:63, AR314:62, AR033:61, AR284:59, AR019:54, AR291:55, AR186:55, AR298:55, AR273:55, AR184:54, AR292:53, AR104:53, AR284:59, AR061:57, AR219:56, AR310:55, AR185:55, AR298:55, AR273:45, AR266:45, AR292:53, AR104:53, AR290:53, AR253:51, AR280:50, AR312:49, AR270:46, AR292:38, AR265:34, AR315:43, AR293:34, AR175:41, AR096:41, AR213:41, AR089:40, AR247:38, AR282:38, AR266:37, AR269:37, AR183:36, AR229:35, AR309:37, AR299:37, AR183:36, AR229:35, AR293:37, AR299:37, AR183:30, AR237:29, AR241:29, AR293:24, AR060:24, AR243:27, AR179:26, AR283:26, AR293:27, AR293:27, AR179:26, AR283:26, AR293:27, AR293:
343	нтени93	722254	353	AR184:5, AR265:4, AR263:3, AR310:2, AR254:2, AR273:2, AR244:2, AR161:2, AR163:2, AR266:2, AR217:2, AR288:2, AR225:1, AR192:1, AR264:1, AR284:1, AR284:1, AR245:1, AR162:1, AR282:1, AR287:1, AR172:1, AR298:1, AR312:1, AR181:1 H0038:3, L0758:3, H0616:1 and L0779:1.

Tissue Distribution Library Code:Count (see Table 4 for Library Codes)		AR162:8, AR161:7, AR163:7, AR229:6, AR183:6, AR176:6, AR173:6, AR313:5, AR178:1, AR240:5, AR162:8, AR162:8, AR163:7, AR233:7, AR229:6, AR184:5, AR182:5, AR274:5, AR166:4, AR221:4, AR275:4, AR175:4, AR263:4, AR263:4, AR262:4, AR270:4, AR175:4, AR264:4, AR300:4, AR282:4, AR268:4, AR261:4, AR267:4, AR267:4, AR267:4, AR282:3, AR282:2, AR282:2, AR282:2, AR282:2, AR282:2, AR282:2, AR282:2, AR282:2, AR282:2, AR282:1, AR382:1, AR282:1, AR2	AR263:33, AR223:32, AR214:31, AR309:30, AR224:29, AR264:29, AR283:27, AR308:27, AR222:27, AR169:25, AR235:25, AR172:25, AR277:24, AR212:23, AR053:23, AR213:22, AR171:22, AR316:31, AR221:31, AR231:21, AR261:21, AR261:21, AR217:24, AR212:23, AR053:19, AR211:21, AR261:21, AR261:21, AR217:24, AR208:20, AR170:20, AR055:19, AR219:19, AR282:19, AR165:19, AR261:21, AR261:11, AR216:19, AR162:19, AR207:18, AR207:18, AR104:18, AR096:17, AR216:19, AR164:18, AR207:18, AR207:18, AR206:15, AR206:11, AR206:1, AR206:1, AR206:1, AR206:1, AR206:1, AR206:1, AR206:1, AR206:1, H0058:2, L0766:2, H0521:2, L0748:2, L0759:1, L0559:1, H0542:1 and H0543:1.	AR235:6, AR215:6, AR242:5, AR162:4, AR161:4, AR192:4, AR165:4, AR053:4, AR163:4, AR269:4, AR164:4, AR291:4, AR288:4, AR166:4, AR166:4, AR271:4, AR282:3, AR236:3, AR217:3, AR264:3, AR261:3, AR178:3, AR270:3, AR177:3, AR272:3, AR1825:3, AR297:3, AR294:3, AR172:3, AR182:3, AR295:3, AR296:3, AR055:3, AR060:3, AR285:3, AR179:3, AR197:3, AR197:3, AR297:3, AR297:3, AR297:3, AR293:3, AR175:3, AR183:3, AR293:3, AR195:3, AR293:3, AR195:3, AR293:3, AR173:2, AR188:2, AR311:2, AR262:2, AR290:2, AR263:2, AR266:2, AR188:2, AR186:2, AR266:2,
SEQ ID NO:X	595	354	355	356
SEQ Contig ID: NO:3	423009	520468	836072	847090
cDNA Clone ID	HTEHU93	HTEIP36	нтвср17	HTELS08
Gene No:		344	345	346

HTEPG70 834931 357 HTHCA18 908144 358 HTHCA18 906536 596 HTJMA95 706618 359	Gene No:	cDNA Clone	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
HTEPG70 834931 357 HTHCA18 908144 358 HTHCA18 906536 596 HTJMA95 706618 359					
HTEPG70 834931 357 HTHCA18 908144 358 HTHCA18 906536 596 HTJMA95 706618 359					AR104:2, AR171:1, AR200:1, AR219:1, AR309:1, AR227:1, AR203:1, AR256:1, AR039:1, AR234:1, AR169:1, AR232:1 H0616:2, L0758:2 and H0038:1.
HTHCA18 908144 358 HTHCA18 906536 596 HTJMA95 706618 359	347	HTEPG70	834931	357	AR176:9, AR282:7, AR162:7, AR161:7, AR163:7, AR055:7, AR182:7, AR060:6, AR266:6, AR253:6, AR201:6, AR228:5, AR242:5, AR242:5, AR246:5, AR2428:5, AR2428:5, AR2428:5, AR2428:5, AR2428:5, AR2428:5, AR248:5, AR248:
HTHCA18 908144 358 HTHCA18 906536 596 HTJMA95 706618 359					AR263:5, AR165:5, AR186:5, AR181:5, AR214:5, AR223:4, AR246:4, AR183:4, AR164:4, AR236:4, AR239:4, AR300-4, AR257:4, AR273:4, AR173:4, AR177:4, AR193:4, AR185:4,
HTHCA18 908144 358 HTHCA18 906536 596 HTJMA95 706618 359					AR230:4, AR089:4, AR218:4, AR277:4, AR179:4, AR192:4, AR264:4, AR039:4, AR237:4, AR104:4, AR316:4,
HTHCA18 908144 358 HTHCA18 906536 596 HTJMA95 706618 359					AR061:4, AR243:4, AR175:4, AR300:4, AR222:4, AR240:4, AR299:4, AR231:4, AR224:3, AR096:3, AR312:3,
HTHCA18 908144 358 HTHCA18 906536 596 HTJMA95 706618 359					AK308:3, AK1/3:3, AK243:3, AK212:3, AK220:3, AK190:3, AK211:3, AK280:3, AK247:3, AK2/4:3, AK213:3, AK213:3, AR288:3, AR288:3, AR174:3, AR197:3, AR191:3, AR296:3, AR207:3, AR221:3, AR221:3, AR2621:3, AR227:3,
HTHCA18 908144 358 HTHCA18 906536 596 HTJMA95 706618 359	-				AR287.3, AR199.3, AR190.3, AR290.3, AR180.3, AR234.3, AR311.2, AR313.2, AR203.2, AR291.2, AR200.2,
HTHCA18 908144 358 HTHCA18 906536 596 HTJMA95 706618 359					AR272:2, AR294:2, AR232:2, AR216:2, AR188:2, AR295:2, AR258:2, AR033:2, AR260:2, AR285:2, AR189:2,
HTHCA18 908144 358 HTHCA18 906536 596 HTJMA95 706618 359					AK297:2, AK171:2, AK203:2, AK193:2, AK119:2, AK108:2, AK210:2, AK213:1, AK230:1, AK172:4, AK111:1, AR235:1, AR235:1, AR169:1 H0616:3, L0758:3, L0717:1, H0038:1 and L0779:1.
HTHCA18 906536 596 HTJMA95 706618 359	348	HTHCA18	908144	358	AR313:12, AR161:10, AR162:10, AR163:10, AR165:8, AR164:8, AR166:8, AR229:7, AR089:7, AR176:7, AR196:7,
HTHCA18 906536 596 HTJMA95 706618 359	?))	AR197:6, AR039:6, AR198:6, AR169:6, AR178:6, AR300:6, AR228:5, AR180:5, AR299:5, AR247:5, AR269:5,
HTHCA18 906536 596 HTJMA95 706618 359					AR182:5, AR309:5, AR193:5, AR233:5, AR204:5, AR282:5, AR212:5, AR239:5, AR200:5, AR207:5, AR267:5,
HTHCA18 906536 596 HTJMA95 706618 359					AR181:5, AR096:5, AR173:5, AR060:5, AR275:4, AR257:4, AR053:4, AR055:4, AR183:4, AR168:4, AR201:4,
HTHCA18 906536 596 HTJMA95 706618 359					AR271:4, AR177:4, AR242:4, AR191:4, AR174:4, AR185:4, AR175:4, AR179:4, AR274:4, AR199:4, AR104:4,
HTHCA18 906536 596 HTJMA95 706618 359					AR23/:4, AR206:4, AR316:4, AR192:4, AR236:4, AR226:4, AR193:4, AR27/:4, AR189:4, AR272:4, AR232:4, App. 1.4, App. 1.
HTHCA18 906536 596 HTJMA95 706618 359					AR231:3, AR203:3, AR293:3, AR230:3, AR262:3, AR234:3, AR258:3, AR270:3, AR289:3, AR296:3, AR227:3,
HTHCA18 906536 596 HTJMA95 706618 359					AR297:3, AR190:3, AR255:3, AR232:3, AR215:3, AR294:3, AR188:3, AR291:3, AR286:2, AR263:2, AR216:2,
HTHCA18 906536 596 HTJMA95 706618 359					AR172:2, AR287:2, AR246:2, AR288:2, AR290:2, AR285:2, AR033:2, AR256:2, AR213:2, AR308:2, AR222:2,
HTHCA18 906536 596 HTJMA95 706618 359					AR218:2, AR224:2, AR223:2, AR311:2, AR283:1, AR211:1, AR219:1, AR295:1, AR221:1, AR170:1, AR260:1,
HTJMA95 706618 359		111111111111	968900	908	
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ARZ43:3, ARZ93:3, ARZ93:3, ARZ92:3, ARZ02:3, ARI00:3, AR309:2, AR393:2, AR392:2, AR300:3, AR3 AD370:3 AD370:3 AD370:3 AD370:3 AD370:3 AR370:3 AR370:	349	HTJMA95	706618	329	
					AK243:3, AK183:3, AK298:3, AK202:3, AK109:3, AK309:2, AK309:2, AK193:2, AK292:2, AK100:2, AK208:2, AK109:2, AK200:2, AK109:2, AK200:2, AK2

Gene No:	cDNA Clone	Contig ID:	SEQ ID	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
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				AR272.2, AR089.2, AR244.2, AR213:1, AR300:1, AR299:1, AR312:1, AR161:1, AR204:1, AR212:1, AR172:1,
				AR198:1, AR285:1, AR061:1, AR288:1, AR293:1, AR176:1, AR275:1, AR241:1, AR163:1, AR247:1, AR174:1,
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				AR237:1, AR225:1, AR258:1, AR259:1 H0264:4, H0488:4, S0328:3, S0306:2, S0476:1, H0272:1, H0487:1,
				H0494:1, H0633:1, L5623:1, L2264:1, L3665:1 and S0330:1.
350	HTJML75	1040047	360	AR060:649, AR055:574, AR089:351, AR104:347, AR299:273, AR185:273, AR283:264, AR039:256, AR277:206,
}				AR282:198, AR316:168, AR096:156, AR300:130, AR240:129, AR313:65, AR219:62, AR218:61, AR169:4, AR170:3,
				AR269:3, AR176:3, AR053:3, AR245:3, AR178:2, AR223:2, AR261:2, AR221:2, AR164:2, AR171:2, AR274:2,
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				AR033:1, AR179:1, AR163:1, AR199:1, AR210:1, AR257:1, AR312:1, AR168:1, AR255:1, AR291:1 H0618:5,
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				H0529:2, L0768:2, L0803:2, H0539:2, L0754:2, L0779:2, L0758:2, H0542:2, S0114:1, H0341:1, H0483:1, S0442:1,
				L0586:1, H0013:1, S0280:1, H0012:1, H0687:1, H0413:1, H0623:1, S0150:1, S0002:1, L0637:1, L0372:1, L0800:1,
				L0643:1, L0644:1, L0645:1, L0774:1, L0809:1, L5623:1, L0666:1, L0663:1, L0664:1, S0052:1, H0519:1, H0690:1,
				\$0328:1, \$0378:1, H0521:1, H0522:1, \$0190:1, H0436:1, \$0037:1, \$0027:1, L0750:1, \$0436:1, L0601:1, L0366:1,
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	CAMILLY	0/00/0	-1	COTOLY COORT COORT COTOLY COTOL
351	HTLAA40	519329	361	AR231:9, AR183:4, AR172:3, AR225:3, AR180:2, AR216:2, AR263:2, AR223:2, AR29:2, AR224:2, AR242:2, AR245:1, AR165:2, AR164:2, AR178:1, AR266:1, AR291:1, AR272:1, AR089:1, AR210:1, AR169:1,
				AR060:1, AR311:1 L0747.2, L0758.2, H0253:1, L0771:1, L0750:1 and L0599:1.
352	HTLEP53	634852	362	AR173:20, AR262:20, AR313:19, AR196:16, AR161:16, AR162:15, AR175:15, AR163:15, AR258:15, AR165:15,
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cDNA Clone		HTLFES7	HTLFE57	HTLFE57	HTLIV19	HTNBO91
Gene No:		353			354	355

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358 HTOHM15 1028538 368 HTOHM15 848200 601 HTOHM15 848200 601 HTOHM15 848196 602 359 HTPBW79 1317835 369	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
HTOHM15 1028538 HTOHM15 848199 HTOHM15 848200 HTOHM15 848200 HTOHM15 1317835	
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HTOHM15 848199 HTOHM15 848200 HTOHM15 848196 HTPBW79 1317835	
HTOHM15 848199 HTOHM15 848200 HTOHM15 848196 HTPBW79 1317835	L0748:12, L0749:8, L0742:1, L0747:1, L0759:0, L0760:0, L0776:0, L0754:0, L0754:0, L0755:4, L0742:4, L0756:3, H0556:3, T0006:3, H0181:3, H0617:3, L0770:3, L0774:3, L0638:3, L0438:3, H0547:3, L0779:3, L0777:3, L0731:3, h0556:3, T0758:3, 10580:3, L0591:3, S0360:2, S0132:2, H0009:2, H0264:2, H0266:2, L0638:2, L0771:2, h056:2, h056:
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HTOHM15 848199 HTOHM15 848200 HTOHM15 848196 HTPBW79 1317835	L0768:1, L0375:1, L0651:1, L0629:1, L0518:1, L0545:1, L0545:1, L0548:1, L0647:1, L0663:1, S0126:1, H0435:1, S0330:1, H0521:1, S0406:1, S3012:1, S3014:1, S0027:1, L0601:1, H0422:1 and H0352:1.
HTOHM15 848200 HTOHM15 848196 HTPBW79 1317835	009
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	HTPBW79	581435	603	
	HTPBW79	396459	604	
360	HTPCS72	854941	370	AR219:14, AR218:13, AR104:11, AR240:9, AR060:8, AR055:7, AR299:7, AR096:7, AR316:7, AR185:6, AR313:6, AR089:6, AR300:6, AR039:5, AR283:4, AR282:4, AR277:2 L0438:6, L0439:5, H0661:3, L0776:3, H0556:2, H0100:2, L0598:2, L0764:2, L0766:2, H0672:2, L0777:2, L0731:2, H0170:1, H0171:1, H0265:1, H0140:1, S0114:1, H0657:1, H0656:1, H0638:1, S0408:1, H0730:1, H0741:1, S0046:1, H0411:1, S0278:1, H0550:1, S0222:1, H0600:1, S0280:1, S0474:1, H0600:1, H064:1, H0640:1, L0640:1, L0640:1, L0640:1, L0640:1, L0640:1, L0640:1, L0770:1, L0
	HTPCS72	266683	605	
361	нтрин83	919916		AR176:5, AR180:5, AR266:5, AR182:5, AR223:4, AR267:4, AR183:4, AR293:4, AR269:4, AR181:4, AR228:4, AR236:4, AR245:4, AR224:4, AR169:3, AR225:3, AR238:3, AR231:3, AR265:3, AR265:3, AR265:4, AR264:4, AR264:4, AR169:3, AR225:3, AR228:3, AR268:3, AR265:3, AR265:3, AR261:3, AR265:3, AR277:3, AR293:3, AR293:3, AR293:3, AR293:3, AR296:3, AR266:3, AR170:3, AR170:3, AR175:3, AR266:3, AR170:3, AR170:3, AR170:3, AR270:2, AR240:2, AR285:2, AR280:2, AR294:2, AR060:2, AR190:2, AR287:2, AR287:2, AR266:2, AR266:2, AR267:2, AR285:2, AR295:2, AR200:2, AR291:2, AR292:2, AR190:2, AR247:2, AR189:2, AR309:2, AR203:2, AR296:2, AR265:1, AR266:1, AR268:1, AR268:1, AR286:1, AR089:1, AR297:1, AR258:1, AR268:1, AR089:3, L0809:3, L0809:2, L0774:2, L0775:2, L0778:2, H0484:1, H0014:1, S0440:1, L0646:1, L0643:1, L0764:1, L0764:1, L0771:1, L0773:1, L0662:1, L0809:1, and L0788:1.
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	HTPIH83	898088	607	
362	HTSEW17	460579	372	AR170:7, AR161:7, AR162:7, AR163:7, AR182:7, AR225:6, AR176:6, AR282:5, AR228:5, AR223:5, AR266:5,

Gene	cDNA Clone	Contig ID:	SEC	Tissue Distribution Library Code: Count
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				AR218:1 H0087:1, S0002:1, L0769:1, L0789:1, H0683:1, H0670:1, L0748:1, L0749:1, L0752:1 and L0758:1.
363	HTTBI76	637725	373	AR252:4, AR214:4, AR309:3, AR169:3, AR297:3, AR193:3, AR250:3, AR271:3, AR291:3, AR161:3, AR272:2,
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·				L0804:1, L0632:1, L0666:1, H0682:1, H0684:1, H0525:1, S0044:1, S0406:1, H0555:1, L0747:1, L0750:1, L0752:1,
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364	HTTBS64	1008159	374	AR282:4, AR252:4, AR269:3, AR171:3, AR170:3, AR264:2, AR176:2, AR291:2, AR311:2, AR225:2, AR277:2, AR168:2, AR270:2, AR172:2, AR262:1, AR271:1, AR055:1, AR272:1, AR299:1, AR257:1, AR313:1 H0040:1
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	HTTBS64	754125	609	
365	HTWCT03	429618		AR096:36, AR218:36, AR219:35, AR039:27, AR283:26, AR089:25, AR282:24, AR316:23, AR313:19, AR299:16,
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	4			AK294:1, AK284:1, AK281:1, AKZ70:1 L0493:4, L2497:1, L0100:1, L0103:1.
366	HTXDW56	695765	376	AR215:23, AR248:19, AR216:19, AR217:16, AR244:15, AR186:14, AR1/0:14, AR197:14, AR025:14, AR096:13, AR254:13 AR36:13 A
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				ANIOZ.11, ANZ.36.11, ANIOS.11, ANZ.14.11, ANZ.25.11, ANZ.16.11, ANZ.14.10, ANZ.14.10, ANZ.14.10, ANZ.16.10, AN
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Gene No:	cDNA Clone ID	Contig ID:		Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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				AR256:8, AR219:8, AR185:8, AR033:8, AR213:8, AR189:8, AR206:8, AR201:8, AR289:8, AR253:8, AR297:8, AR214:9, AR263:8, AR210:8, AR263:8, AR291:9, AR241:9, AR2
				AR211:7, AR274:7, AR193:7, AR188:7, AR222:7, AR299:7, AR262:7, AR206:7, AR206:7,
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				AR281:2, AR252:1 S04/4:17, L0803:16, L0/48:13, S0408:11, L2609:11, L204:10, L0/70:10, L0803:9, L0/34:9, S0402:8, 1.0809:7, S0360:5, L0794:5, L0755:5, L0731:5, L0758:5, H0265:4, S0414:4, H0581:4, H0046:4, H0009:4,
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				H0586:2, H0069:2, H0123:2, H0622:2, H0031:2, H0644:2, H0616:2, H0551:2, L0598:2, L0766:2, L0655:2, L0659:2,
				L0636:2, L0664:2, L0665:2, H0144:2, S0374:2, H0547:2, H0660:2, S0378:2, H0436:2, L0750:2, L0756:2, H0624:1, L0040:1, H0484:1, E0440:1, E0440:1, E0440:1, E0440:1, H0480:1, H0484:1, E0440:1, E0440:1, E0440:1, E0440:1, H0480:1, H0480:1, E0440:1, E0440:1, E0440:1, E0440:1, H0480:1, H0480:1, H0480:1, E0440:1, E0440:1, E0440:1, E0440:1, H0480:1, H0480:1, H0480:1, E0440:1, E0440:1, E0440:1, E0440:1, H0480:1, H0480:1, E0440:1, E04
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367	HTXJM03	603918	377	AR313:13, AR252:10, AR282:8, AR312:7, AR176:7, AR096:7, AR269:6, AR254:6, AR201:6, AR196:6, AR245:6,
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				ARZ3513, ARZ0113, ARZ1113, ARZ0313, ARITO013, ARX11211, ARX10313, ARZ1013, ARZ1013, ARZ1013, ARZ1013, ARZ1013,

Gene	cDNA Clone	SEQ ID ID ID IN	SEQ ID	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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368	HTXON32	838288	378	AR195:107, AR197:91, AR172:81, AR246:78, AR295:74, AR272:72, AR258:71, AR196:67, AR224:67, AR235:67, AR171:66, AR193:66, AR291:63, AR297:59, AR223:58, AR168:57, AR200:56, AR263:55, AR222:54, AR170:53,
A,				AR261:53, AR245:52, AR236:52, AR169:52, AR311:49, AR256:49, AR225:49, AR188:48, AR173:48, AK285:48, AR285:48, AR288:47, AR221:46, AR260:46, AR198:46, AR313:46, AR174:45, AR201:45, AR271:45, AR191:44, AR175:44,
				AR217:44, AR286:44, AR287:43, AR309:43, AR270:43, AR264:42, AR211:42, AR274:42, AR308:41, AR199:41,
				AR033:38, AR255:37, AR296:37, AR210:36, AR190:36, AR257:36, AR289:35, AR213:35, AR282:34, AR240:34,
				AR218:34, AR163:32, AR247:32, AR176:31, AR180:30, AR312:30, AR254:30, AR212:30, AR166:29, AR300:29,
				AR162:29, AR293:29, AR203:29, AR183:29, AR219:28, AR161:28, AR192:28, AR242:28, AR165:27, AR250:27, AR550:27, AR56:27, AR182:28, AR182:23, AR182:2
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369	HUDBESS	1177561	6/6	AR264:3, AR162:3, AR161:3, AR169:3, AR271:3, AR195:3, AR195:3, AR277:3, AR164:3, AR253:3, AR207:3,
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				L0601:2, S0218:1, H0650:1, H0656:1, H0254:1, H0255:1, H0327:1, H0070:1, H0040:1, T0042:1, L0809:1, L0790:1, L0792:1, H0689:1, H0435:1, H0660:1, H0134:1, L0741:1, L0759:1 and S0042:1.
	HUDBZ89	562791	910	
370	HUFBY15	1352349	1	AR310:36, AR309:31, AR312:30, AR052:28, AR265:24, AR213:15, AR273:14, AR249:13, AR263:12, AR313:12, AR251:12, AR248:12, AR274:10, AR053:10, AR315:10, AR253:9, AR280:8, AR314:8, AR219:7, AR096:6,

HUFBY15 846380 HUFCJ30 638402 HUKAH51 1352424	SEQ 11 381 381 381 382	Tissue Distribution Library Codes) AR218:6, AR089:6, AR299:6, AR316:5, AR192:5, AR271:5, AR186:4, AR039:4, AR282:4, AR206:4, AR244:3, AR059:3, AR206:3, AR200:3, AR300:3, AR317:2, AR246:2, AR329:4, AR329:3, AR327:2, AR329:3, AR3
+	┿	50550C.1, 30140C.1, file+ (6.1, 30020.1 and 110425.1.
HUKAH51 130	1300737 612	
H	603538 613	

Gene	cDNA Clone	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code:Count (see Table 4 for Library Codes)
373	HUKBT29	694590		
374	HUSXS50	1352367	384	AR253:15, AR270:12, AR184:11, AR268:11, AR263:11, AR262:11, AR182:10, AR096:10, AR060:10, AR248:10, AR219:9, AR269:9, AR313:8, AR238:8, AR290:8, AR240:8, AR240:8, AR240:10, AR269:9, AR313:8, AR238:8, AR290:8, AR240:8, AR240:8, AR240:10, AR269:7, AR291:7,
	HUSXS50	883176	614	
	HUSXS50	655372	615	
375	HUVEB53	571200	385	AR053:3, AR171:3, AR224:3, AR180:2, AR168:2, AR207:2, AR165:2, AR282:2, AR217:2, AR299:2, AR234:1, AR277:1, AR296:1, AR295:1, AR164:1, AR266:1, AR205:1, AR265:1, AR269:1, AR269:1,

Gene No:	cDNA Clone	Contig ID:	SEQ ID NO:X	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
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376	HVARW53	1194812	386	H0423:1. AR055:226, AR060:204, AR240:159, AR299:129, AR185:119, AR300:117, AR283:117, AR039:107, AR277:104, AR104:102, AR282:101, AR089:73, AR316:65, AR096:53, AR313:37, AR218:32, AR219:29 S0378:3, S0380:3, L4558:1 and H0764:1.
	HVARW53	1044491	919	
377	HWAAD63	838626	387	AR196:17, AR173:14, AR161:14, AR162:14, AR241:14, AR163:14, AR165:13, AR313:12, AR166:12, AR164:12, AR262:12, AR264:11, AR236:11, AR199:10, AR191:10, AR174:9, AR178:9, AR257:9, AR235:9, AR180:9, AR262:12, AR264:11, AR236:11, AR200:8, AR229:8, AR274:7, AR189:7, AR311:7, AR346:7, AR380:9, AR263:8, AR175:7, AR312:7, AR312:7, AR308:7, AR308:7, AR261:7, AR169:7, AR265:7, AR188:7, AR234:6, AR177:6, AR297:7, AR312:7, AR197:7, AR308:7, AR206:5, AR206:5, AR176:6, AR293:6, AR206:5, AR168:6, AR221:6, AR194:6, AR297:6, AR271:6, AR297:7, AR300:5, AR206:5, AR206:3, AR206:1, AR206:1, AR184:1, AR286:1, AR286:1, AR286:1, AR286:1, AR286:1, AR065:1, AR286:1, AR065:1, AR0
	HWAAD63	833089	617	
	HWAAD63	793875	618	
378		580889	388	AR253:12, AR215:9, AR213:8, AR254:7, AR225:7, AR2221:7, AR053:6, AR223:6, AR212:6, AR291:5, AR282:5, AR165:5, AR164:5, AR295:5, AR096:5, AR196:5, AR165:5, AR164:5, AR164:5, AR290:4, AR178:4, AR169:4, AR089:4, AR192:4, AR224:4, AR183:4, AR263:4, AR296:4, AR313:4, AR297:4, AR285:4, AR255:4, AR261:4, AR216:4, AR309:4, AR200:4, AR172:4, AR257:4, AR270:4, AR268:4, AR193:4, AR262:4, AR300:3, AR269:3, AR275:3, AR175:3, AR277:3, AR191:3, AR240:3, AR286:3, AR247:3, AR193:4, AR269:3, AR269:3, AR271:3, AR271:8:3, AR286:3, AR247:3, AR286:3, AR286:3, AR247:3, AR286:3,
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Gene	cDNA Clone		1	Tissue Distribution Library Code: Count
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				AR163:2, AR272:2, AR211:2, AR104:1, AR283:1, AR242:1, AR252:1, AR245:1, AR256:1, AR176:1 H0581:2
370	HWAD189	799506	389	AR252:29, AR250:29, AR253:21, AR254:10, AR282:6, AR215:6, AR165:5, AR164:5, AR166:5, AR089:5, AR161:5,
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380	HWBAR88	836469	390	AR241.5, AR263.4, AR268.3, AR197.3, AR214.3, AR252.3, AR249.3, AR193.2, AR162.2, AR166.2, AR161.2,
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381	HWBCB89	1093347	391	AR207:18, AR222:18, AR283:17, AR223:17, AR214:17, AR263:16, AR224:16, AR169:16, AR089:15, AR316:14,
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				L0520:1, L0763:1, L0769:1, L4556:1, L0806:1, L0805:1, L0647:1, L0789:1, L0663:1, H0144:1, S3012:1, L0748:1, L0749:1, L0731:1, L0757:1, H0653:1, H0543:1, H0423:1 and H0352:1.
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382	HWBCP79	846382	392	AR313:44, AR039:36, AR196:28, AR089:27, AR096:25, AR299:23, AR300:21, AR185:18, AR163:17, AR161:17, AR167:17 AR240:16 AR277:15 AR164:15 AR218:15 AR173:14, AR316:14, AR165:14, AR229:13, AR199:13,
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			<u></u>	AR311:3, AR207:3, AR225:3, AR224:3, AR289:2, AR232:2, AR168:2, AR23:2, AR214:2, AR266:2, AR222:2, AR111:3, AR254:3, AR172:1, AR061:1, H0580:1, and H0169:1.
	HWBCP79	646977	620	
383	HWBDP28	1352265	393	AR271:15, AR163:11, AR060:11, AR161:11, AR162:11, AR165:10, AR254:10, AR164:10, AR166:9, AR263:9,
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				AR311:8, AR2/2:1, AR190:1, AR282:1, AR2/0:1, AR1/3:1, AR1/0:1, AR240:1, AR230:1, AR333:1, AR313:1, AR323:1, AR323:1, AR323:1, AR303:1, A
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				H0580:1, H0486:1, T0039:1, H0575:1, S6028:1, H0130:1, S0002:1, L0769:1, L0646:1, L0783:1, L0790:1, L0666:1,

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Gene No:	cDNA Clone	Contig ID:	A S	Tissue Distribution Library Code: Count (see Table 4 for Library Codes)
				L0665:1, H0435:1, H0696:1, L0749:1 and H0506:1.
	HWBDP28	638536	621	
384	HWBFX31	799427		AR171:3, AR309:2, AR271:2, AR225:2, AR205:2, AR267:2, AR213:2, AR257:2, AR236:2, AR053:1, AR266:1, AR179:1, AR199:1, AR270:1, AR214:1, AR181:1, AR240:1, AR247:1, AR247:1, L0659:5, L0794:4, L0809:4, L0777:4, H0424:3, L0766:3, L0745:3, H0265:2, H0656:2, H0656:2, H0662:2, S0376:2, H0457:2, H0024:2, L0768:2, H0670:2, H055:2, L0751:2, L0780:2, H0556:1, H0218:1, H0224:1, H0638:1, S0360:1, H06457:1, H06457:1, H0688:1, H0580:1, H0688:1, H0675:1, H0063:1, H0063:1, H0087:1, H0564:1, H0772:1, H0652:1, S0002:1, S0426:1, L0763:1, L0770:1, L0760:1, L0773:1, L0648:1, L0762:1, L0748:1, L0773:1, H0522:1, L0773:1, L0762:1, L0752:1, L0759:1, L0750:1, L0
385	HWHHL34	805642	395	AR266:56, AR291:51, AR292:48, AR292:44, AR295:34, AR270:32, AR218:23, AR218:23, AR218:23, AR291:51, AR292:48, AR292:44, AR293:23, AR218:23, AR310:23, AR319:21, AR283:13, AR292:02, AR295:19, AR096:18, AR316:18, AR039:17, AR285:17, AR285:13, AR299:16, AR309:16, AR309:16, AR309:16, AR299:10, AR299:10, AR299:13, AR299:14, L0770:14, L069:14, L0770:14, L069:14, L0770:14, L069:14, L0770:14, L069:14, L0770:14, L0770:14, L069:14, L0770:14, L0770:14, L069:14, L0770:14, L0770:14, L069:14, L0770:14, L0770:14, L079:14, L0779:14, L07

Gene No:	cDNA Clone Contig ID:	Contig ID:	SEQ NO:X	
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				L0653:1, L0606:1, L0657:1, L0656:1, L0517:1, L0384:1, L0544:1, L0788:1, L0791:1, L0792:1, L0663:1, S0053:1, S0374:1, S0148:1, H0519:1, S0126:1, S0330:1, S0380:1, H0710:1, H0518:1, H0525:1, H0696:1, S0044:1, S0390:1, S3014:1, S0206:1, L0786:1, L0780:1, L0755:1, L0686:1, H0665:1, S0196:1 and H0506:1.
	HWHHL34	801943	622	
	HWHHL34	341560	623	
386	HWHQS55	762842	396	
				AR163:18, AR172:17, AR311:17, AR216:17, AR309:17, AR313:17, AR291:17, AR254:17, AR308:17, AR165:16,
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				ARI/1:13, AR103:13, AR108:13, AR1/0:13, AR222:13, AR212:13, AR2/0:12, AR175:10, AR210:10, AR178:10,
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				AR262:8, AR267:8, AR190:8, AR296:8, AR177:8, AR235:8, AR225:8, AR174:8, AR053:8, AR300:8, AR257:8,
				AR211:8, AR055:8, AR197:8, AR219:8, AR185:8, AR218:8, AR199:8, AR060:7, AR236:7, AR195:7, AR287:7,
				AR261:7, AR193:7, AR215:7, AR061:7, AR295:7, AR258:7, AR285:7, AR196:7, AR192:6, AR191:6, AR256:6,
				AR204:6, AR104:6, AR207:6, AR207:6, AR294:6, AR201:6, AR182:6, AR286:6, AR231:5, AR198:5, AR200:5,
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				AR230:3, AR226:3, AR228:3 L0439:6, H0620:4, L0758:2, S0040:1, S0282:1, H0661:1, H0619:1, H0549:1, H0587:1,
				H0013:1, L0021:1, H0230:1, H0009:1, H0373:1, H0135:1, L0770:1, L0769:1, L0776:1, L0659:1, L0783:1, H0144:1,
0	JAM HILL	2002	255	H0519:1, H0593:1, H0682:1, H0659:1, L0/51:1, L0/53:1 and L0/59:1.
38/	сонтжи	(1/66/	160	ARVOLLS), ARCS 1.00, ARCS 6.00, ARCS 7.30, ARCS 4.31, ARCS 9.31, ARS 14:30, ARCS 8.26, ARCS 7.32, ARCS 84:23.
				AR283:22, AR265:22, AR266:21, AR310:20, AR263:19, AR292:19, AR033:18, AR298:17, AR184:17, AR192:17,
				AR246:17, AR243:17, AR096:17, AR233:15, AR295:15, AR177:15, AR282:15, AR198:13, AR186:13, AR267:13,
				AR299:13, AR273:12, AR316:12, AR104:12, AR296:12, AR251:12, AR291:12, AR249:12, AR247:12, AR219:12,
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				AR218:10, AR259:10, AR286:10, AR268:10, AR269:10, AR204:10, AR055:9, AR182:9, AR270:9, AR253:9,
				AR175:8, AR183:8, AR309:8, AR053:8, AR312:8, AR271:8, AR089:7, AR275:7, AR294:7, AR185:7, AR256:7,
				AR290:7, AR274:7, AR293:6, AR258:6, AR060:5, AR109:3, AR160:3, AR161:3, AR162:3, AR264:3, AR163:3,

Gene	cDNA Clone	Contig ID:	SEQ ID NO:X	
			-	L0596:2, S0040:1, H0294:1, S0430:1, H0656:1, S0358:1, S0360:1, H0729:1, H0645:1, H0586:1, H0587:1, H0632:1, H0590:1, L0045:1, S0003:1, H0316:1, H0598:1, S0036:1, H0591:1, L0564:1, H0560:1, H0509:1, H0641:1, S0002:1,
				L0640:1, L0662:1, L0775:1, L0655:1, L0659:1, L0783:1, L5622:1, L0663:1, L2653:1, H0701:1, H0689:1, H0672:1, H0539:1, S0406:1, L0439:1, L0749:1, L0786:1, S0434:1, S0436:1, H0543:1, S0424:1 and S0446:1.
388	HYAAJ71	826754	398	AR313:41, AR173:25, AR163:25, AR166:25, AR196:23, AR161:23, AR162:23, AR165:22, AR164:21, AR089:21, AR313:30, AR318:19, AR318:18, AR374:18, AR174:18, AR175:18, AR191:17,
				ARISSIT, AR262:17, AR308:17, AR257:16, AR275:16, AR229:16, AR247:16, AR309:16, AR199:16, AR179:15,
				AR189:13, AR183:13, AR240:13, AR270:13, AR000:14, AR203:13, AR033:13, AR192:13, AR177:13, AR219:13,
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				AR285:11, AR182:11, AR242:11, AR261:11, AR212:11, AR29/:11, AR188:11, AR190:11, AR203:11, AR260:10, AP260:10,
				AR2537.9, AR252.9, AR250.9, AR271.9, AR198.9, AR195.9, AR039.9, AR230.9, AR211.9, AR266.8, AR231.8,
				AR289.8, AR210.8, AR245.8, AR272.8, AR201.8, AR197.8, AR277.7, AR239.7, AR227.7, AR207.7, AR283.7,
				AR253:7, AR267:7, AR228:7, AR235:7, AR254:7, AR204:7, AR232:6, AR243:6, AR205:6, AR250:6, AR061:5,
				AR055:5, AR246:5, AR172:4, AR168:4, AR223:4, AR222:3, AR170:3, AR217:3, AR217:3, AR171:2, AR225:2,
				AR215:1, AR224:1, AR221:1 H0583:1, H0485:1, H0581:1, S0053:1 and H0423:1.
389	HYBAR01	610383	399	AR308:42, AR192:7, AR205:4, AR161:3, AR198:3, AR178:3, AR162:3, AR163:3, AR193:3, AR206:3, AR169:3,
				ARI 10:3, ARZ 10:3, ARU89:3, ARZ 40:3, ARZ 40:3, ARZ 41:3, ARZ 41:3, ARU 40:3, ARI 40:3, ARI 40:3, ARZ 40:
				AR282.2. AR275.2. AR262.2. AR264.2, AR213.2, AR288.2, AR104.2, AR272.1, AR182.1, AR225.1, AR183:1,
				AR166:1, AR311:1, AR294:1, AR165:1, AR299:1, AR283:1, AR229:1, AR181:1, AR312:1, AR217:1 H0041:1,
				L0471:1 and L0766:1.
390	HYBBE75	834784	400	AR215:6, AR252:4, AR162:4, AR161:4, AR163:4, AR183:3, AR309:3, AR165:3, AR164:3, AR176:3, AR235:3, AR165:3, AR270:3 AR204:3, AR245:3, AR196:3, AR196:2, AR196:2, AR242:2, AR257:2, AR277:2, AR196:2,
				AR089.2, AR201.2, AR250.2, AR266.2, AR313.2, AR182.2, AR291.2, AR255.2, AR233.2, AR060.2, AR282.2,
				AR225:2, AR197:2, AR214:2, AR239:2, AR247:2, AR294:2, AR185:2, AR293:2, AR268:2, AR285:2, AR177:2,
				AR213:2, AR287:2, AR178:2, AR237:1, AR174:1, AR230:1, AR267:1, AR316:1, AR240:1, AR181:1, AR096:1,
				[AR228:1, AR290:1, AR286:1, AR232:1, AR296:1, AR286:1, AR189:1, AR061:1, AR221:1, AR289:1, AR220:1,
				ARI/9:1, ARZ38:1, ARZ95:1, AR300:1, AR300:1, AR310:1

SEQ Tissue Distribution Library Code:Count (see Table 4 for Library Codes)	401 AR186:8, AR310:7, AR274:6, AR033:6, AR218:5, AR313:5, AR104:5, AR219:5, AR202:5, AR226:5, AR039:4, AR055:4, AR183:4, AR246:4, AR184:4, AR238:3, AR192:3, AR167:3, AR163:3, AR247:3, AR183:4, AR248:4, AR284:4, AR288:3, AR275:3, AR282:3, AR275:3, AR282:3, AR275:3, AR282:3, AR275:3, AR282:3, AR282:2, AR269:2, AR269:2, AR266:2, AR267:2, AR277:2, AR277:2, AR312:2, AR291:2, AR295:1, AR292:1, AR295:1,	624	625
S GI S	7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7	Н	┢
Contig	846517	887467	878627
Gene cDNA Clone Contig ID: NO:X	HAPSA79	HAPSA79	HAPSA79
Gene No:	391		

Table 1C summarizes additional polynucleotides encompassed by the invention (including cDNA clones related to the sequences (Clone ID:), contig sequences (contig identifier (Contig ID:) contig nucleotide sequence identifiers (SEQ ID NO:X)), and genomic sequences (SEQ ID NO:B). The first column provides a unique clone identifier, "Clone ID:", for a cDNA clone related to each contig sequence. The second column provides the sequence identifier, "SEQ ID NO:X", for each contig sequence. The third column provides a unique contig identifier, "Contig ID:" for each contig sequence. The fourth column, provides a BAC identifier "BAC ID NO:A" for the BAC clone referenced in the corresponding row of the table. The fifth column provides the nucleotide sequence identifier, "SEQ ID NO:B" for a fragment of the BAC clone identified in column four of the corresponding row of the table. The sixth column, "Exon From-To", provides the location (i.e., nucleotide position numbers) within the polynucleotide sequence of SEQ ID NO:B which delineate certain polynucleotides of the invention that are also exemplary members of polynucleotide sequences that encode polypeptides of the invention (e.g., polypeptides containing amino acid sequences encoded by the polynucleotide sequences delineated in column six, and fragments and variants thereof).

TABLE 1C

5

10

15

cDNA Clone	SEQ ID	CONTIG ID:	BAC ID: A	SEQ ID	EXON
ID	NO:X			NO:B	From-To
HAIBP89	25	727543	AC005214	1241 ·	1-228
					817-3471
HAIBP89	25	727543	AC005214	1242	1-539
HAUAI83	42	639009	AC010422	1243	1-326
					1552-2084
					2162-2261
					2300-2427
					4485-5868
			!		5948-6362
					7914-8017
:					8569-8681
					8765-8875
				ļ	8968-9037
					9284-9499
ł					9742-9910
				Į.	10837-11187
			•		11271-11321
				1	11554-11707
					11783-12766
					12866-13225
					13256-13827
					14284-14367
					14890-15090
HAUAI83	42	639009	AC018761	1244	1-326
					1176-1284
					1552-2084

cDNA Clone	SEQ ID	CONTIG ID:	BAC ID: A	SEQ ID	EXON
ID	NO:X			NO:B	From-To
					2162-2261
					2300-2426
			:		4485-5868
					5948-6362
					8569-8681
					8765-8875
		1			8968-9037
		1			9284-9499
					9742-9910
					10317-10501
					10837-11187
					11271-11321
					11554-11707
					11783-12766
			·		12866-13225
			,		13256-13827
					14284-14367
					14890-15090
HAUAI83	42	639009	AC010422	1245	1-315
					2004-2289
Ì					2650-2741
					3554-3830
HAUAI83	42	639009	AC010422	1246	1-202
					938-1047
				•	1219-1395
]	1758-1956
·					2907-3429
					3792-3935
					5366-5485
					6391-6688
				l	6899-7269
					7890-8316
					8400-8524
			1		8607-8682
1					8824-8999
					9190-9302
					9691-9796
					10106-10177
					10571-11051
					11164-11490 12565-12696
					13364-13501
					13964-14592
					14740-15540
					15610-15798
					15947-16642
					16717-16832
				1	16968-17408
					17521-17612
					18331-18579
	1			1	19120-19303
					19358-19514
	<u></u>	<u> </u>	<u> </u>	<u> </u>	19599-19702

cDNA Clone	SEQ ID	CONTIG ID:	BAC ID: A	SEQ ID	EXON
ID	NO:X_	<u> </u>		NO:B	From-To_
				10.5	20003-20245
HAUAI83	42	639009	AC018761	1247	1-202
					938-1047
					1219-1395
					1758-1956
					2907-3429
					3792-3935
					5366-5485
					6391-6688
		,]	6899-7269
1					7591-7711
				1	7890-8316
					8400-8524
					8607-8682 8749-9073
					9190-9302
					9691-9796
	40	620000	A CO19761	1248	1-82
HAUAI83	42	639009	AC018761	1248	128-293
					1178-1447
					1986-2278
.					2457-2711
		·			3543-3844
TID CDD22	46	1252402	AC024191	1249	1-643
HBCPB32	46	1352403	AC024191	1249	1421-1636
·					4917-5536
HBCQL32	47	1134954	AC069250	1250	1-461
HBCQL32	47	1134934	AC009230	1230	504-1011
					1964-2424
					2747-2859
					3098-3251
l i					4239-6717
HBCQL32	47	1134954	AC069250	1251	1-418
HBINS58	51	1352386	AL096774	1252	1-1023
122.000	0.2				2010-2239
					2581-2962
					3153-3223
			1		3324-3493
					3973-4126
HBINS58	51	1352386	AL096774	1253	1-341
HBINS58	51	1352386	AL096774	1254	1-142
HBMCI50	55	668268	AL139132	1255	1-890
HBMCI50	55	668268	AL359179	1256	1-891
HBMCI50	55	668268	AL139132	1257	1-155
HBMCI50	55	668268	AL359179	1258	1-155
HCE3G69	61	728432	AC068946	1259	1-108
					1186-1324
					1746-1835
					2138-2284
					2448-2545
					2718-2861
				1	3091-5889

cDNA Clone	SEQ ID	CONTIG ID:	BAC ID: A	SEQ ID	EXON
ID	NO:X		<u></u>	NO:B	From-To
HCE3G69	61	728432	AC068946	1260	1-191
HCE3G69	61	728432	AC068946	1261	1-686
HCEFB80	65	1143407	AL022327	1262	1-2271
					3506-3658
			:		4643-4810
1					9039-9164
					9382-9509
					10587-10720
					11135-11195
					11265-11716
					14644-15466
					17451-17526
					18012-18114
					20530-20632
					20957-21009
					23696-23785
					25338-25575
					25969-26166
HCEWE17	67	941941	AL139130	1263	1-170
					463-598
					623-1346
]				ı	1404-1523
1					2059-2159
					2350-2616
					3068-3254
					3428-3878
HCNDR47	71	1016919	AL122003	1264	1-236
					531-696
					787-817
					863-4508
		1			5158-5744
770170717		1016010	AT 100000	1265	6949-7029
HCNDR47	71	1016919	AL122003	1265	1-888
					1304-2003 2830-3284
					3719-4571
		,			4618-5268
			·		6131-6557
					8947-9033
			}		9058-9726
					14176-14480
					18456-18915
				ļ	18960-19871
				1	22365-22454
]				1	23082-23248
					28058-28215
HCOOS80	73	1134974	AC003688	1266	1-718
11000300	,,,	1134714	ACOOSOG		1054-1158
					1660-1980
					4003-4073
					4364-4516
				-	4646-4749
	L	<u> </u>	L	L	1 1010-1172

cDNA Clone	SEQ ID	CONTIG ID:	BAC ID: A	SEQ ID	EXON
ID	NO:X	[NO:B	From-To
					4852-4995
					5121-5213
					5354-5424
					5526-5669
!					5759-5832
					5850-6176
					6756-6829
					7023-7175
					7259-7398
		1			7531-7711
					8134-8381
1				i	8463-13585
					13691-14323
1					14437-14918
HCOOS80	73	1134974	AC026954	1267	1-138
					273-453
				1	876-1123
					1205-4456
HCOOS80	73	1134974	AC003688	1268	1-125
1100000			110000		203-480
					1463-1647
		į			2048-2077
					2229-2323
					2725-3784
					3867-4682
HDPGT01	89	771583	AC020978	1269	1-180
					2776-2899
					3916-4077
					4296-4335
					4436-4632
					4895-5181
				İ	8153-8246
		1			9547-9666
		:			9907-10007
	1				10370-10618
					10788-11046
					11926-13423
					13465-13494
					13764-15689
HDPGT01	89	771583	AC020978	1270	1-384
HDPSB18	98	1043263	AL355512	1271	1-2572
					3049-3871
HDPSB18	98	1043263	AC006176	1272	1-2571
		1			3048-3872
HDPSB18	98	1043263	AL355512	1273	1-280
HDPWN93	104	992925	AC004590	1274	1-276
					489-591
					866-988
					1106-1281
					1323-1444
				1	1632-1799
1					1866-2016
			<u> </u>	<u></u>	1000 2010

cDNA Clone	SEQ ID	CONTIG ID:	BAC ID: A	SEQ ID	EXON
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					2109-2313
					2634-3205
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					3528-3744
			•		3820-5006
					6580-6919
					7076-7276
					8057-8153
					8318-8680
HDPWN93	104	992925	AC021491	1275	1-275
TIDE WINSO	104	772723	ACOZITA	1275	488-590
		1			865-987
					1105-1280
					1322-1443
		<u> </u>			1631-1798
					1865-2015
		,			2108-2312
·					2633-3204
				•	3359-3471
					3527-3743
					3819-5005
					6579-6918
					1 1
					7075-7275
ļ					8054-8150
			1.500.1500	1076	8315-8677
HDPWN93	104	992925	AC004590	1276	1-303
					727-1252
					5721-5846
HDPWN93	104	992925	AC021491	1277	1-303
					727-1253
					5723-5848
HDPXY01	105	879048	AL354000	1278	1-1319
					4848-4975
İ					5229-5600
		<u> </u>			6561-6654
HDPXY01	105	879048	AL035362	1279	1-1316
		-			4844-4971
		-			5225-5596
					6557-6650
HDPXY01	105	879048	AL354000	1280	1-460
HDPXY01	105	879048	AL354000	1281	1-400
HDPXY01	105	879048 ,	AL035362	1282	1-400
HDPXY01	105	879048	AL035362	1283	1-460
HDTFE17	111	1043391	AF196972	1284	1-74
			1		391-524
			ļ	1	1481-1536
	İ				1623-1699
				1	2092-2448
] .		2537-2611
	1	1			3085-3179
1	1	1			3315-3395
1					6429-6514
L			L	<u> </u>	1 0127-0314

cDNA Clone	SEQ ID	CONTIG ID:	BAC ID: A	SEQ ID	EXON
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					7611-7693
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					9534-9680
		ļ			9770-9875
					10373-10876
HDTFE17	111	1043391	AF196972	1285	1-742
HFIIZ70	140	1043350	AC005005	1286	1-368
					1579-2971
HFIIZ70	140	1043350	AC005005	1287	1-484
					517-1142
]			2842-3176
		[3376-3493
İ					3575-3740
					3873-4227
, ' l					4728-4935
		1			5074-5351
			·	·	5446-5564
					5772-5960
		}			7287-7627
					7721-8097
					8218-9325 12098-12161
					12098-12161
		ľ			13482-13666
		1			13748-13817
.			:		14445-14519
					14595-14928
					15658-15754
					15848-15923
					16016-16112
					16512-16660
:				ļ	21313-21448
					21710-21870
					21899-22470
1				·	22634-22787
					23169-23307
HFOXA73	143	850699	AC005866	1288	1-523
HFOXA73	143	850699	AC007618	1289	1-522
HHENK42	162	493724	AC023105	1290	1-192
					355-585
]					1654-1995
					3493-3802
					3827-4082
<u> </u>					5266-5952
					6109-6292
					6819-6947
					7118-8308
				1	8602-8887
					9337-9517
					10052-10284
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cDNA Clone	SEQ ID	CONTIG ID:	BAC ID: A	SEQ ID	EXON
ID	NO:X			NO:B	From-To
HHENK42	162	493724	AC023105	1291	1-286
HHENK42	162	493724	AC023105	1292	1-754
HHGCG53	166	340818	AC024037	1293	1-518
HHGCM76	167	662329	AC003665	1294	1-70
					304-609
					900-1090
		}			1240-1835
					2272-2490
					2581-3598
HHGCM76	167	662329	AC003665	1295	1-580
					851-995
					1224-1296
			!		1314-1663
			,		1930-1975
		1			2724-2905
					2968-3098
					3283-3328
				·	5121-5230
777 1 6 6 6 6 6	1674	205505	A C010512	1206	5331-5689
HJACG30	174	895505	AC018512	1296 1297	1-776 1-878
HJACG30	174	895505	AC022305		1-150
HJACG30	174	895505	AC002518	1298 1299	1-130
HLTIP94	207	1087335	AC007431	1300	1-330
HLTIP94	207	1087335	AC007431 AC012086	1300	1-3328
HMSDL37	229	973996		1302	1-3328
HMSDL37	229 229	973996 973996	AC018811 AC018494	1303	1-3031
HMSDL37	229	973996	AC018494 AC012086	1304	1-224
HMSDL37	229	973996	AC012086	1305	1-468
HMSDL37	229	973996	AC012080 AC018811	1306	1-222
HMSDL37	229	973996	AC018811	1307	1-468
HMSDL37	229	973996	AC018494	1308	1-224
HMSDL37	229	973996	AC018494	1309	1-1854
HNGIH43	249	410179	AC018980	1310	1-83
пиошчэ	243	410179	ACO10700	1510	3147-4045
	•	İ			4401-4443
HNGIH43	249	410179	AC018977	1311	1-604
HNGIH43	249	410179	AL356243	1312	1-83
Intonis		120275	122555		3146-4044
					4400-4442
HNGIH43	249	410179	AC018980	1313	1-872
HNGOI12	256	1041375	AC003675	1314	1-2128
HNGOI12	256	1041375	AC001228	1315	1-2129
HNGOI12	256	1041375	AC013791	1316	1-2132
HNHFM14	260	664507	AC020552	1317	1-290
HNHFM14	260	664507	AC020552	1318	1-96
HPJBK12	293	1011467	AC022033	1319	1-2649
НРЈВК12	293	1011467	AC013541	1320	1-2649
HPJBK12	293	1011467	AC022033	1321	1-190
HPJBK12	293	1011467	AC013541	1322	1-190
HPRAL78	295	1352342	AC007783	1323	1-2334
III ICIDIO		1 1000042	1 110001100	1	

cDNA Clone	SEQ ID	CONTIG ID:	BAC ID: A	SEQ ID	EXON
· ID	NO:X			NO:B	From-To
					2508-3084
					3578-3890
					4198-4294
				:	4376-4623
					4712-5349
					5369-6088
					6527-7107
					7298-7392
					7730-7846
					9147-9476
					10487-10575
					10791-11298
					11485-11601
					11783-13009
					13207-13501
					13540-13772
					14439-14800
					14923-14983
					15133-15355
•					15485-15653
	1			ł	16750-16805
					16894-17078
					17162-17219
					18003-18089
	ĺ				21085-21146
					21358-21501
HPRAL78	295	1352342	AC007783	1324	1-308
HPRAL78	295	1352342	AC007783	1325	1-1024
HPWAY46	300	1001560	AC019036	1326	1-1399
HPWAY46	300	1001560	AC067828	1327	1-1399
HPWAY46	300	1001560	AC019036	1328	1-788
HPWAY46	300	1001560	AC067828	1329	1-788
HRGBL78	308	910133	AL359541	1330	1-254
	:				2777-3307
					3670-3823
			1		4113-4385
					4844-5381
				1221	5995-7365
HSAWD74	313	460527	AC004951	1331	1-1651
				1222	1740-2593
HSAWD74	313	460527	AC004951	1332	1-149
HSAWD74	313	460527	AC004951	1333	1-5057
	1				5082-8353
	ļ		1,5005	1004	8404-8996
HSDJL42	321	1036471	AC008676	1334	1-56
				 	571-2959
HSLJG37	331	1016920	AC022608	1335	1-2406
HSLJG37	331	1016920	AC022608	1336	1-53
					430-718
HSLJG37	331	1016920	AC022608	1337	1-351
HTHCA18	358	908144	AP002439	1338	1-1800
HTHCA18	358	908144	AP002505	1339	1-1776

cDNA Clone	SEQ ID	CONTIG ID:	BAC ID: A	SEQ ID	EXON
ID	NO:X			NO:B	From-To
HTHCA18	358	908144	AP002439	1340	1-110
HTHCA18	358	908144	AP002505	1341	1-110
HTJML75	360	1040047	AC025036	1342	1-148
HTJML75	360	1040047	AC022232	1343	1-152
HTJML75	360	1040047	AC022231	1344	1-151
HTJML75	360	1040047	AC010694	1345	1-202
HTJML75	360	1040047	AC027300	1346	1-158
HTJML75	360	1040047	AC011953	1347	1-126
HTJML75	360	1040047	AC010694	1348	1-77
HTLIV19	364	1046341	AC055750	1349	1-964
HTLIV19	364	1046341	AC027463	1350	1-964
HTLIV19	364	1046341	AC055750	1351	1-236
HTLIV19	364	1046341	AC027463	1352	1-236
HTPCS72	370	854941	AL008639	1353	1-106
					1457-1595
Ì	,			•	1666-2484
					2910-3006
				•	3705-4147
j					4768-5141
					5304-5536
					5746-5874
					7114-7241
					7468-7711
					7963-8746
					9438-12408
					12884-14976
HTPCS72	370	854941	AL008639	1354	1-720
HTPIH83	371	919916	AL158821	1355	1-1862
				1	1880-3126
HVARW53	386	1194812	AC011298	1356	1-648
					1184-3022
		1			3943-4047
			1 6011060	1057	5961-6504
HVARW53	386	1194812	AC011298	1357	1-397

Table 1D: The polynucleotides or polypeptides, or agonists or antagonists of the present invention can be used in assays to test for one or more biological activities. If these polynucleotides and polypeptides do exhibit activity in a particular assay, it is likely that these molecules may be involved in the diseases associated with the biological activity. Thus, the polynucleotides or polypeptides, or agonists or antagonists could be used to treat the associated disease.

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The present invention encompasses methods of detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating a disease or disorder. In preferred embodiments, the present invention encompasses a method of treating a hematopoietic and hematologic diseases and disorders comprising administering to a patient in which such detection, treatment, prevention,

and/or amelioration is desired a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof) in an amount effective to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate the hematopoietic and hematologic diseases and disorder.

In another embodiment, the present invention also encompasses methods of detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating a hematopoietic and hematologic diseases and disorder; comprising administering to a patient combinations of the proteins, nucleic acids, or antibodies of the invention (or fragments or variants thereof), sharing similar indications as shown in the corresponding rows in Column 3 of Table 1D.

Table 1D provides information related to biological activities for polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof). Table 1D also provides information related to assays which may be used to test polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof) for the corresponding biological activities. The first column ("Gene No.") provides the gene number in the application for each clone identifier. The second column ("cDNA Clone ID:") provides the unique clone identifier for each clone as previously described and indicated in Table 1A through Table 1D. The third column ("AA SEQ ID NO:Y") indicates the Sequence Listing SEQ ID Number for polypeptide sequences encoded by the corresponding cDNA clones (also as indicated in Tables 1A, Table 1B, and Table 2). The fourth column ("Biological Activity") indicates a biological activity corresponding to the indicated polypeptides (or polynucleotides encoding said polypeptides). The fifth column ("Exemplary Activity Assay") further describes the corresponding biological activity and also provides information pertaining to the various types of assays which may be performed to test, demonstrate, or quantify the corresponding biological activity.

Table 1D describes the use of, inter alia, FMAT technology for testing or demonstrating various biological activities. Fluorometric microvolume assay technology (FMAT) is a fluorescence-based system which provides a means to perform nonradioactive cell- and bead-based assays to detect activation of cell signal transduction pathways. This technology was designed specifically for ligand binding and immunological assays. Using this technology, fluorescent cells or beads at the bottom of the well are detected as localized areas of concentrated fluorescence using a data processing system. Unbound flurophore comprising the background signal is ignored, allowing for a wide variety of homogeneous assays. FMAT technology may be used for peptide ligand binding assays, immunofluorescence, apoptosis, cytotoxicity, and bead-based immunocapture assays. See, Miraglia S et. al., "Homogeneous cell and bead based assays for highthroughput screening using flourometric microvolume assay technology," Journal of Biomolecular Screening; 4:193-204 (1999). In particular, FMAT technology may be used to test, confirm, and/or identify the ability of polypeptides (including polypeptide fragments and variants) to activate signal transduction pathways. For example, FMAT technology may be used to test, confirm, and/or identify the ability of polypeptides to upregulate production of immunomodulatory

proteins (such as, for example, interleukins, GM-CSF, Rantes, and Tumor Necrosis factors, as well as other cellular regulators (e.g. insulin)).

Table 1D also describes the use of kinase assays for testing, demonstrating, or quantifying biological activity. In this regard, the phosphorylation and de-phosphorylation of specific amino acid residues (e.g. Tyrosine, Serine, Threonine) on cell-signal transduction proteins provides a fast, reversible means for activation and de-activation of cellular signal transduction pathways. Moreover, cell signal transduction via phosphorylation/de-phosphorylation is crucial to the regulation of a wide variety of cellular processes (e.g. proliferation, differentiation, migration, apoptosis, etc.). Accordingly, kinase assays provide a powerful tool useful for testing, confirming, and/or identifying polypeptides (including polypeptide fragments and variants) that mediate cell signal transduction events via protein phosphorylation. See e.g., Forrer, P., Tamaskovic R., and Jaussi, R. "Enzyme-Linked Immunosorbent Assay for Measurement of JNK, ERK, and p38 Kinase Activities" Biol. Chem. 379(8-9): 1101-1110 (1998).

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TABLE 1D

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B ₹	Biological Activity Ex	Exemplary Activity Assay
Φ. IJ II. (N)	Stimulation of Assinsular secretion mo from pancreatic ant beta cells. upp in city ago 277 PE Par AT AT AT AT AT AT ASSINGLE COLUMN ASSINGLE CELL COLUMN ASSINGLE CELL CELL CELL CELL CELL CELL CELL C	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
CHITTE	Activation of As transcription knot through serum invesponse element response element in immune cells trail (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Vints Genes 12(2):107,105,117 (1997), the content of each of which are herein incompared

			by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.
Activation of Signaling Pa in immune co (such as eosinophils).	1 T QL '00'	thway ells	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Bio Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochen Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reactions. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang IP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of -Jun NHZ-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils." Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74 (1999); the contents of each, "In vivo resistance to orticosteroids in bronchial asthma is associated with enhanc
Production of TNF alpha by dendritic cells	S 4 5	t of t by ells	TNFa FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells, fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and cytotoxic effects on a variety of cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, modulate inflammation and cytotoxicity. Exemplary assays that test for immunomodulatory proteins evaluate the production of

				cytokines such as tumor necrosis factor alpha (TNFa), and the induction or inhibition of an inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Verhasselt et al., Eur J Immunol 28(11):3886-3890 (1198); Dahlen et al., J Immunol 160(7):3585-3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.
c	H6EAB28	628	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
4	Н6ЕDС19	629	Regulation of viability and proliferation of	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of

			pancreatic beta	pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the
			cells.	number of viable cells in culture based on quantitation of the ATP present which signals the presence of metaholically active cells. Exemplary assays that may be used or routinely modified to
	•			test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention
	-			(including antibodies and agonists or antagonists of the invention) include assays disclosed in:
				Friedrichsen Biv, et al., Mol Endocrinol, 13(1):130-46 (2001), ruddat 1924, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the
				contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that
				may be used according to these assays are publicly available (e.g., through the ATCC) and/or may
				be routinely generated. Exemplary pancreatic cells that may be used according to these assays
				include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated
				from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of
				native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al.
				Endocrinology 1992 130:167.
8	H6EDF66	630	Activation of	Assays for the activation of transcription through the AP1 response element are well-known in the
	•		transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through AP1	(including antibodies and agonists or antagonists of the invention) to modulate growth and other cell
			response element	functions. Exemplary assays for transcription through the AP1 response element that may be used
			in immune cells	or routinely modified to test AP1-response element activity of polypeptides of the invention
			such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem
				272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al.,
				Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by
				reference in its entirety. Human T cells that may be used according to these assays are publicly
		_		available (e.g., through the ATCC). Exemplary human T cells that may be used according to these
_				assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.
S	H6EDF66	630	Production of IL-	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used
			10 and activation	or routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			of T-cells.	agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation
				of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of
				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
		_		modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed
				and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-

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				968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL.13, IL.5 and IL.6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	×
9	H6EDX46	631	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "Th-elper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	TL D. S.
7	НАСВD91	632	Activation of transcription through cAMP response element (CRE) in preadipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in	ប្

				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Preadipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
7	НАСВD91	632	Activation of transcription through cAMP response element in immune cells (such as T-cells).	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP and regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is a suspension culture of IL-2 dependent cytotoxic T cells.
7	HACBD91	632	Production of IL-6	IL-6 FMAT. IL-6 is produced by T cells and has strong effects on B cells. IL-6 participates in IL-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6 induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and differentiation and modulate T cell proliferation and function. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and functional activities. Such assays that may be used or

routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipoccytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ilpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatomytes that may be used according to these assays includes the H4IIE rat liver hepatoma cell line.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48
	Regulation of transcription of Malic Enzyme in adipocytes	Activation of Endothelial Cell p38 or JNK Signaling Pathway.
	632	632
	HACBD91	HACBD91
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				(1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis,
7	HACBD91	632		Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.
	HACBD91	632	Activation of transcription through AP1 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the AP1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the AP1 response element that may be used or routinely modified to test AP1-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.

7	HACBD91	632	Activation of	Assays for the activation of transcription through the CD28 response element are well-known in the
			through CD28	art and may be used of fournery moduled to assess the ability of purypeptities of the invention (including antibodies and agonists of antigonists of the invention) to stimulate IL-2 expression in T
			response element	cells. Exemplary assays for transcription through the CD28 response element that may be used or
			in immune cells	routinely modified to test CD28-response element activity of polypeptides of the invention
			(such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol
-,				159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J
				Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference
			_	in its entirety. T cells that may be used according to these assays are publicly available (e.g.,
				through the ATCC). Exemplary human T cells that may be used according to these assays include
				the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
7	HACBD91	632	Activation of	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			transcription	response element are well-known in the art and may be used or routinely modified to assess the
			through NFAT	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			response element	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			in immune cells	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
			(such as T-cells).	element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66.1-10 (1998); Cullen and Malm, Methods in
·		_		Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);
				Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol
				31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J
<u> </u>				Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by
				reference in its entirety. T cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human T cells that may be used according to these assays
				include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
7	HACBD91	632	Activation of	Assays for the activation of transcription through the Signal Transducers and Activators of
			transcription	Transcription (STAT6) response element are well-known in the art and may be used or routinely
			through STAT6	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
			response element	antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of
			in immune cells	multiple genes. Exemplary assays for transcription through the STAT6 response element that may
			(such as T-cells).	be used or routinely modified to test STAT6 response element activity of the polypeptides of the

invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biochem 26(2) and Cham 26(2) and Cham 26(2) and Cham 26(2) and Cham 26(2) and Proprients of each of which are herein incorporated by
	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).
	632	632
	НАСВ D 91	HACBD91
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×	HACCI17	633	Production of IL-8	Assay that measures the production of the chemokine interleukin-8 (IL -8) from immine cells (such
)		}		as the EOL-1 human eosinophil cell line) are well known in the art (for example, measurement of
			(such as the	IL-8 production by FMAT) and may be used or routinely modified to assess the ability of
			human EOL-1	polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to
			eosinophil cells)	promote or inhibit. Eosinophils are a type of immune cell important in allergic responses; they are
				recruited to tissues and mediate the inflammtory response of late stage allergic reaction. IL8 is a
				strong immunomodulator and may have a potential prointlammatory role in immunological diseases
~	HACC117	633	Activation of	This renotter assay measures activation of the GATA-3 signaling nathway in HMC-1 human mast
• 		3		cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
			through GATA-3	production. Assays for the activation of transcription through the GATA3 response element are
			response element	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
			in immune cells	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
			(such as mast	transcription factors and modulate expression of mast cell genes important for immune response
			cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test GATA3-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
				mast cells.
∞	HACCI17	633	Activation of	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell
			transcription	line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production.
			through NFAT	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
4.			response element	response element are well-known in the art and may be used or routinely modified to assess the
			in immune cells	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			(such as mast	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response

				polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
∞	HACCI17	633	Production of IL-5	IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.
∞	HACCI17	633	Production of VCAM in endothelial cells	Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to

			(such as human umbilical vein	meaure the upregulation of cell surface VCAM-1 expresssion in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to,
			endothelial cells (HUVEC))	angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein
		_ _		endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106) a membrane associated protein can be unreculated by cytokines or other factors.
				and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood
				vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.
∞	HACCI17	633	Production of IL-8 by by endothelial	Assays measuring production of IL-8 are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
	-		cells (such as	antagonists of the invention) to regulate production and/or secretion of IL-8. For example, FMAT
			Human Umbilical	may be used or routinely modified to assess the ability of polypeptides of the invention (including
			Cord Endothelial	antibodies and agonists or antagonists of the invention) to regulate production and/or secretion of
			Cells).	are endothelial cells which line venous blood vessels and are involved in functions that include but
		_		are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell
		_		extravasation. Endothelial cells play a pivotal role in the initiation and perpetuation of inflammation
				and secretion of IL-8 may play an important role in recruitment and activation of immune cells such
				as neutrophils, macrophages, and lymphocytes.
8	HACCI17	633	Production of	Endothelial cells, which are cells that line blood vessels, and are involved in functions that include,
			ICAM in	but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell
			endothelial cells	extravasation. Exemplary endothelial cells that may be used in ICAM production assays include
			(such as human	human umbilical vein endothelial cells (HUVEC), and are available from commercial sources. The
			umbilical vein	expression of ICAM (CD54),a intergral membrane protein, can be upregulated by cytokines or other
			endothelial cells	factors, and ICAM expression is important in mediating immune and endothelial cell interactions
			(HUVEC))	leading to immune and inflammatory responses. Assays for measuring expression of ICAM-1 are
				well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
				the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1
				expression. Exemplary assays that may be used or routinely modified to measure ICAM-1
				expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000);
				Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J
				Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein
				incorporated by reference in its entirety.
6	HAGAI85	634	Production of	IFNgamma FMAT. IFNg plays a central role in the immune system and is considered to be a

			IFNgamma using	proinflammatory cytokine. IFNg promotes TH1 and inhibits TH2; promotes IgG2a and inhibits
			Natural Killer	IgE; induces macrophage activation; and increases MHC expression. Assays for
			ceiis	inflammatory activities and inhibit TH2 helper cell functions are well known in the art and may be
				used or routinely modified to assess the ability of polypeptides of the invention (including
				antibodies and agonists or antagonists of the invention) to mediate immunomodulation, regulate
				inflammatory activities, modulate TH2 helper cell function, and/or mediate humoral or cell-
				mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the
				production of cytokines, such as Interferon gamma (IFNg), and the activation of T cells. Such
				assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of
				the invention (including antibodies and agonists or antagonists of the invention) include the assays
				disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al.,
				"Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal
				8(5):225-233 (1995); Billiau et al., Ann NY Acad Sci 856:22-32 (1998); Boehm et al., Annu Rev
				Immunol 15:749-795 (1997), and Rheumatology (Oxford) 38(3):214-20 (1999), the contents of each
				of which are herein incorporated by reference in its entirety. Natural Killer (NK) cells that may be
		_		used according to these assays are publicly available (e.g., through the ATCC) or may be isolated
		_		using techniques disclosed herein or otherwise known in the art. Natural killer (NK) cells are large
				granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-
				independent killing of tumor cells and also recognize antibody bound on target cells, via NK Fc
				receptors, leading to cell-mediated cytotoxicity.
6	HAGA185	634	Production of	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and
		=	GM-CSF	fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage
				progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage.
				Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and
				monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory
				cytokine. Assays for immunomodulatory proteins that promote the production of GM-CSF are well
				known in the art and may be used or routinely modified to assess the ability of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) to mediate
				immunomodulation and modulate the growth and differentiation of leukocytes. Exemplary assays
				that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF,
				and the activation of T cells. Such assays that may be used or routinely modified to test
				immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular

				Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Ye et al., J Leukoc Biol (58(2):225-233, the contents of each of which are herein
				assays are publicly available (e.g., through the ATCC) or may be isolated using to incoed assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclosed herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor
				cells and also recognize antibody bound on target cells, via INN FC receptors, leading to cell-mediated cytotoxicity.
01	HAGAQ26	635	Stimulation of insulin secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is
			beta cells.	measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is
				upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component
	-			in diabetes. Exemplary assays that may be used of founding mounded to test for similariting of incoming exemplary from nancreatic cells) by polymentides of the invention (including antibodies and
				answill secretion (1101) painerealie eeus) by posperiores of the invention (1101) and possess and
				277(4 Pt 2):R959-66 (1999); Li. M. et al., Endocrinology, 138(9):3735-40 (1997); Kim. K.H., et al.,
				FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-
_				204 (1999), the contents of each of which is herein incorporated by reference in its entirety.
				Pancreatic cells that may be used according to these assays are publicly available (e.g., through the
				ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according
				to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from
				cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics
				typical of native pancreatic beta cells including glucose inducible insulin secretion. References:
=	HAGDI35	636	Activation of	Assaur et al. Entroclinology 1992 190.10/. Assaur for the activation of transcription through the NEKR response element are well-known in the
;		8		art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through NFKB	(including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription
			response element	factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription
			in immune cells	through the NFKB response element that may be used or rountinely modified to test NFKB-
			(such as EOL1	response element activity of polypeptides of the invention (including antibodies and agonists or
			cells).	antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen
				and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA
				83:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J

				Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. For example, a reporter assay (which massings in transcription inducible from a NFRR responsive element in FOL-1 cells) may
				link the NFKB element to a repeorter gene and binds to the NFKB transcription factor, which is
				these assays include eosinophils such as the human EOL-1 cell line of eosinophils. Bosinophils are
				a type of immune cell important in the allergic responses; they are recruited to tissues and mediate the inflammtory response of late stage allergic reaction. Bol-1 is a human eosinophil cell line.
11	HAGDI35	636	Activation of	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast
			transcription	cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
			through GATA-3	production. Assays for the activation of transcription through the GATA3 response element are
			response element	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
			in immune cells	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
			(such as mast	transcription factors and modulate expression of mast cell genes important for immune response
			cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test GATA3-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
				mast cells.
11	HAGDI35	989	Production of	Assays for measuring expression of VCAM are well-known in the art and may be used or routinely
			VCAM in	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
			endothelial cells	antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to
			(such as human	meaure the upregulation of cell surface VCAM-1 expresssion in endothelial cells. Endothelial cells
			umbilical vein	are cells that line blood vessels, and are involved in functions that include, but are not limited to,
			endothelial cells	angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary
			(HUVEC))	endothelial cells that may be used according to these assays include human umbilical vein

	·			endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors, and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.	rs, od
	HAGDI35	636	Production of IL-8 by by endothelial cells (such as Human Umbilical Cord Endothelial Cells).	Assays measuring production of IL-8 are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate production and/or secretion of IL-8. For example, FMAT may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate production and/or secretion of IL-8 from endothelial cells (such as human umbilical vein endothelial cells (HUVEC)). HUVECs are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Endothelial cells play a pivotal role in the initiation and perpetuation of inflammation and secretion of IL-8 may play an important role in recruitment and activation of immune cells such as neutrophils, macrophages, and lymphocytes.	or E E S S but but uch
11	HAGDI35	636	Production of ICAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Endothelial cells, which are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used in ICAM production assays include human umbilical vein endothelial cells (HUVEC), and are available from commercial sources. The expression of ICAM (CD54),a intergral membrane protein, can be upregulated by cytokines or other factors, and ICAM expression is important in mediating immune and endothelial cell interactions leading to immune and inflammatory responses. Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety.	de, e C'he rther s re re S of M-1
11	HAGDI3S	929	Activation of transcription through NFKB response element in immune cells	This reporter assay measures activation of the NFkB signaling pathway in Ku812 human basophil cell line. Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for	or se iii

-		(such as basophils).	transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Online and Malm. Methods in Braymol 216:362-368 (1992): Henthorn et al., Proc Natl Acad Sci
			USA 85:6342-6346 (1988); Marone et al, Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Basophils that may be
			used according to these assays are publicly available (e.g., through the ATCC). Exemplary human basophil cell lines that may be used according to these assays include Ku812, originally established
			from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that can be induced to differentiate into mature basophils.
1	637	Regulation of	Assays for the regulation of transcription through the DMEF1 response element are well-known in
		transcription via	the art and may be used or routinely modified to assess the ability of polypeptides of the invention
		element in	element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin
		adipocytes and	production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2
		pre-adipocytes	transcription factor and another transcription factor that is required for insulin regulation of Glut4
			expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat
			and inductive tissue, exemplarly assays that may be used of fortunely incurred to test for Entrangate response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention
			(including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai,
			M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 275(21):16323-8
			(2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair
			regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in
			transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988);
			and, Cullen, B., et al., Methods in Enzymol. 210:362–368 (1992), the contents of each of which is
			nerein incorporated by reference in its entirety. Autpocytes and pre-autpocytes that may be used according to these accove are multicly available (e.g., through the ATCC) and/or may be routinely
			generated. Exemplary cells that may be used according to these assays include the mouse 3T3-L1
			cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous
			substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte
			to adipose-like conversion under appropriate differentiation culture conditions.
L	638	Activation of	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast
		transcription	cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
		through GATA-3	production. Assays for the activation of transcription through the CALA3 response element are

			response element in immune cells	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
			(such as mast cells).	transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test GA1A3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992): Henhorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988): Flavell et al., Cold
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incomorated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
				mast cells.
14	HAIBO71	639	Endothelial Cell	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or
			Apoptosis	routinely modified to assess the ability of polypeptides of the invention (including antibodies and
				agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Induction
				of apoptosis in endothelial cells supporting the vasculature of tumors is associated with tumor
	•			regression due to loss of tumor blood supply. Exemplary assays for caspase apoptosis that may be
				used or routinely modified to test capase apoptosis activity of polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include the assays disclosed in
				Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and
				Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are
				herein incorporated by reference in its entirety. Endothelial cells that may be used according to
				these assays are publicly available (e.g., through commercial sources). Exemplary endothelial cells
				that may be used according to these assays include bovine aortic endothelial cells (bAEC), which
				are an example of endothelial cells which line blood vessels and are involved in functions that
				include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell
				extravasation.
14	HAIB071	639	Activation of	Assays for the activation of transcription through the Signal Transducers and Activators of
			transcription	Transcription (STAT6) response element are well-known in the art and may be used or routinely
			through STAT6	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or

the STAT6 response element that may nt activity of the polypeptides of the fithe invention) include assays falm, Methods in Enzymol 216:362-12-6346 (1988); Georas et al., Blood (7):1521-1523 (2000); Curiel et al., Eur of Chem 275(38):29331-29337 (2000), rence in its entirety. T cells that may through the ATCC). Exemplary rat s are publicly available (e.g., through	r Factor of Activated T cells (NFAT) or routinely modified to assess the and agonists or antagonists of the atte expression of genes involved in iption through the NFAT response l-response element activity of ists or antagonists of the invention) s); Cullen and Malm, Methods in ad Sci USA 85:6342-6346 (1988); r et al., Int J Biochem Cell Biol 3:838-844 (1999); and Yeseen et al., J is of which are herein incorporated by g to these assays are publicly available may be used according to these assays cell line with cytolytic and cytotoxic	s, macrophages, endothelial cells, and ion of granulocytes- macrophage. Is, monocytes and macrophage. nitiation of dendritic cells and considered to be a proinflammatory
antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary rat the ATCC).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage. Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory
response element a in immune cells cach as natural killer cells).	Activation of transcription through NFAT aresponse element in immune cells (such as natural killer cells).	Production of GM-CSF
	639	640
	HABO71	HAIBP89
	4	15

known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and modulate the growth and differentiation of leukocytes. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Ye et al., J Leukoc Biol (58(2):225-233, the contents of each of which are herein incorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclosed herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor cells and also recognize antibody bound on target cells, via NK Fc receptors, leading to cell-mediated cytotoxicity.	IFNgamma FMAT. IFNg plays a central role in the immune system and is considered to be a proinflammatory cytokine. IFNg promotes TH1 and inhibits TH2 differentiation; promotes IgG2a and inhibits IgE secretion; induces macrophage activation; and increases MHC expression. Assays for immunomodulatory proteins produced by T cells and NK cells that regulate a variety of inflammatory activities and inhibit TH2 helper cell functions are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, regulate inflammatory activities, modulate TH2 helper cell function, and/or mediate humoral or cellmediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as Interferon gamma (IFNg), and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal 8(5):225-233 (1995); Billiau et al., Ann NY Acad Sci 856:22-32 (1998); Boehm et al., Annu Rev Immunol 15:749-795 (1997), and Rheumatology (Oxford) 38(3):214-20 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T
	Production of IFNgamma using a T cells
	640
	HAIBP89
	15

				Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.
16	HAICP19	641	Bone marrow cell proliferation (fibronectin enhanced)	Assay for measuring regulation of proliferation of mouse bone marrow cells (in the presence or absence of exogenous Stem Cell Factor (SCF)) on a fibronectin extracllular matrix. Mouse bone marrow cells are plated onto 96-well fibronectin fragment coated plates in 0.2 ml of serum-free medium. Secreted protein factors (test factors) are tested with appropriate negative controls in the presence and absence of SCF (5.0 ng/ml), where secreted test factor supernates represent 10% of the total assay volume. The cells are grown for 7 days. The number of proliferating cells within the wells is quantitated by measuring thymidine incorporation into cellular DNA. This and similar assays may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate proliferation of bone marrow cells. Interactions between adhesion receptors on progenitor cells and their extracellular matrix ligands are essential for the control of hematopoiesis in bone marrow stroma. These interactions may help retain CD34+ hematopoietic progenitor cells within the an appropriate bone marrow environment, and adhesive interactions can also provide important costimulatory signals. As the ability of stem cells to undergo self-renewal in vitro is dependent upon their interaction with the stromal cells and the extracellular matrix (ECM), this assay identify factors which integrate with the ECM environment and are important for stimulating stem cell self-renewal.
16	HAICP19	641	Activation of Adipocyte PI3 Kinase Signalling Pathway	Kinase assay. Kinase assays, for example an GSK-3 assays, for PI3 kinase signal transduction that regulate glucose metabolism and cell survival are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit glucose metabolism and cell survival. Exemplary assays for PI3 kinase activity that may be used or routinely modified to test PI3 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Nikoulina et al., Diabetes 49(2):263-271 (2000); and Schreyer et al., Diabetes 48(8):1662-1666 (1999), the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
16	HAICP19	641	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or

				antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000), Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include Aortic Smooth Muscle Cells (AOSMC): such as bovine AOSMC.
16	HAICP19	641	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1998); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
16	HAICP19	4	Activation of Natural Killer Cell ERK Signaling Pathway.	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-

contents of each of which are herein incorporated by reference in its entirety. Natural killer cells
that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary natural killer cells that may be used according to these assays include the human natural
killer cell lines (for example, NK-YT cells which have cytolytic and cytotoxic activity) or primary NK cells.
642 Stimulation of Assays for measuring calcium flux are well-known in the art and may be used or routinely
compared to much higher extracellular calcium. Extracellular factors can cause an influx of
calcium, leading to activation of calcium responsive signaling pathways and alterations in cell
functions. Exemplary assays that may be used or routinely modified to measure calcium flux by
polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995);Mogami H, et
41., Enforcement (1757) 130(7).2700-0 (1757), Michaluson 3D, et al., Diochem 7, 200 (11.3).047-31
(1992); and, ivieats, JE, et al., Cell Calcium 1969 Ivov-Dec; 10(6):535-41 (1969), the contents of
each of which is nerein incorporated by reference in its entitlety. Fancteatic cells that may be used
generated. Exemplary pancreatic cells that may be used according to these assays include HITT15
Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells
transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors.
The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by
somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219:
643 Regulation of Assays for the regulation of transcription through the PEPCK promoter are well-known in the art
transcription
_
ter
PEPCK promoter activity (in hepatocytes) of polypeptides of the invention (including antibodies
and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10
(1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl

			Acad Sci USA 83:6342-6346 (1988); Lochhead et al., Diabetes 49(b):896-903 (2000); and Yeagley et al., J Biol Chem 275(23):17814-17820 (2000), the contents of each of which is herein incorporated by reference in its entirety. Hepatocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary liver hepatoma cells that may be used according to these assays include H4lle cells, which contain a tyrosine amino transferase that is inducible with glucocorticoids, insulin, or cAMP derivatives.
HAJBR69		Production of GM-CSF	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage. Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory cytokine. Assays for immunomodulatory proteins that promote the production of GM-CSF are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and modulate the growth and differentiation of leukocytes. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Ye et al., J Lewco Biol (\$8(2):225-233, the contents of each of which are herein incorporated by reference in its entirety. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor cells and also recognize antibody bound on target cells, via NK Fc receptors, leading to cell-mediated cytotoxicity.
HAJBZ75	644	Activation of transcription through GAS response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the

				invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).
19	HAJB275	644	Activation of transcription through NFAT response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in inmunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
20	HAMFC93	645	Production of IL- 13 and activation of T-cells.	Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science;282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors

				that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
21	HAMFE15	646	Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed inThai, M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, E., et al., J Biol Chem, 273(23):14285-24 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is an adherent mouse preadipocyte cell line. Mouse 373-L1 cells are a continuous substrain of 373 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.
22	HAMFK58	647	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
23	HAMGR28	648	Stimulation of	Assays for measuring calcium flux are well-known in the art and may be used or routinely

modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium esponsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995); Mogami H, et al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element activity of polypeptides of the used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
Calcium Flux in pancreatic beta cells.	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).
	649
·	HANGG89
	24

			include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	
HANGG89	649	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	
HAPOM49	650	Regulation of viability and proliferation of pancreatic beta cells.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may	

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Exemplar INS-1 cel rat transplacells includ 30:167.	I p38 kinas are well k are well k les of the i or inhibit it in inhibit in of the inve of the inve 998); Gupt 11(3) I Biol 71(3), through the CTLL	issays, for on or diffe on or diffe thion) to pr SRK kinase of polype or polype or polype sec Symp Prog Biop orated by ys are pub y be used a
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be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9): 1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Mouse adjocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse adjpocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under
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	Activation o Cell p38 or J Signaling Pathway.	Activation of Adipocyte ERK Signaling Pathway
	651	652
	HAPPW30	HAPUC89
		H
	26	27

				appropriate differentiation conditions known in the art.
27	HAPUC89	652	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1998); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the FIMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
27	HAPUC89	652	Activation of Skeletal Muscle Cell ERK Signalling Pathway	Kinase assay. Kinase assays, for examplek Elk-1 kinase assays, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9): 1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Rat myoblast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary rat myoblast cells that may be used according to these assays include L6 cells. L6 is an adherent rat myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuses to form multinucleated

				mortuhes and striated fibers after culture in differentiation media
28	HATAC53	653	Production of	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and
2		}		fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage
				progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage.
				Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and
				monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory
				cytokine. Assays for immunomodulatory proteins that promote the production of GM-CSF are well
				known in the art and may be used or routinely modified to assess the ability of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) to mediate
				immunomodulation and modulate the growth and differentiation of leukocytes. Exemplary assays
				that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF,
				and the activation of T cells. Such assays that may be used or routinely modified to test
				immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular
				Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-
				160 (2000); and Ye et al., I Leukoc Biol (58(2):225-233, the contents of each of which are herein
				incomparated by reference in its entirety. Natural killer cells that may be used according to these
				assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclosed
				herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes that
				have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor
				reals and also recognize antibody bound on target cells, via NK Pc recentors, leading to cell-
				mediated cytotoxicity,
29	HATBR65	654	Production of IL-6	L-6 FMAT. L-6 is produced by T cells and has strong effects on B cells. L-6 participates in L-4
				induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6
				induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease,
				plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for
				immunomodulatory and differentiation factor proteins produced by a large variety of cells where the
				expression level is strongly regulated by cytokines, growth factors, and hormones are well known in
				the art and may be used or routinely modified to assess the ability of polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) to mediate immunomodulation
				and differentiation and modulate T cell proliferation and function. Exemplary assays that test for
	-			immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation
				and upregulation of T cell proliferation and functional activities. Such assays that may be used or
				routinely modified to test immunomodulatory and diffferentiation activity of polypeptides of the

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invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipoocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatoma cell line.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
	Regulation of transcription of Malic Enzyme in adipocytes	Production of IL- 10 and activation of T-cells.
	654	655
	HATBR65	HATDF29
	29	30

			
reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL 4, IL 10, IL 13, IL 5 and IL 6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB 1, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is
	Activation of Endothelial Cell p38 or JNK Signaling Pathway.	Production of ICAM-1	Insulin Secretion
	656	959	657
	HATEE46	HATEE46	HAUA183
	31	31	32

ي پ	<u> </u>
upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells stransformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cill proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils." Emplay by intric oxide in exceptor signaline by wirrie oxide in exceptor signaline by wirrie oxide in exceptor signaline by wirrie oxide in exceptor signaline by wirrie.
	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).
	859
	HBAFJ33
	33

				Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of
				each of which are herein incorporated by reference in its entirety.
33	HBAFJ33	929	Upregulation of CD152 and	CD152 FMAT. CD152 (a.k.a. CTLA-4) expression is restricted to activated T cells. CD152 is a negative regulator of T cell proliferation. Reduced CD152 expression has been linked to
			activation of T	hyperproliferative and autoimmune diseases. Overexpression of CD152 may lead to impaired
			cells	immunoresponses. Assays for immunomodulatory proteins important in the maintenance of T cell
				nomeostasts and expressed atmost exclusively on CD4+ and CD6+ I cells are wen known in the arrange may be used or routinely modified to assess the ability of polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) to modulate the activation of T
				cells, maintain T cell homeostasis, and/or mediate humoral or cell-mediated immunity. Exemplary
				assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers,
				such as CD152, and the activation of T cells. Such assays that may be used or routinely modified to
				test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists
				or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J
				Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach"
				Chapter 6:138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al.,
				Curr Opin Immunol 11(3):294-300 (1999); and Saito T, Curr Opin Immunol 10(3):313-321 (1998),
				the contents of each of which are herein incorporated by reference in its entirety. Human T cells
				that may be used according to these assays may be isolated using techniques disclosed herein or
				otherwise known in the art. Human T cells are primary human lymphocytes that mature in the
				thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-
			-	mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory
3.4	HRAEV10	650	Activation of INK	IdCOON. Kinasa assay INK binasa assays for signal transduction that ramilate cell proliferation activation
<u>.</u>		``		or apoptosis are well known in the art and may be used or routinely modified to assess the ability of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to
			(such as	promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase
			eosinophils).	activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides
			-	of the invention (including antibodies and agonists or antagonists of the invention) include the
				assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res
				247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin,

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				Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of	S
46	HBAFV19	629	Upregulation of CD152 and activation of T cells	Coll 52 FMAT. CD152 (a.k.a. CTLA-4) expression is restricted to activated T cells. CD152 is a negative regulator of T cell proliferation. Reduced CD152 expression has been linked to hyperproliferative and autoimmune diseases. Overexpression of CD152 may lead to impaired immunoresponses. Assays for immunomodulatory proteins important in the maintenance of T cell homeostasis and expressed almost exclusively on CD4+ and CD8+ T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate the activation of T cells, maintain T cell homeostasis, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers, such as CD152, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Curr Opin Immunol 11(3):294-300 (1999); and Saito T, Curr Opin Immunol 11(3):294-300 (1999); and Saito T, Curr Opin Immunol 10(3):313-321 (1998), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the	E . 0

				thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory
35	HBAMB15	099	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
36	HBCPB32	661	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays

				include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.
37	нвсос.	662	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
37	нвсог.32	662	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et

al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):1771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of
	Regulation of viability and proliferation of pancreatic beta cells.	Insulin Secretion
	663	664
	HBGBA69	HBIAE26
	38	33

				each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
				generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells
				transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors.
		-		The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by
				somatostatin or giucocorticolus. A11C# CML-1/7/ Nets. Lord and Asheron: Diochem. 3, 213. 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
40	HBIMB51	665	Activation of JNK	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation,
			Signaling Pathway	or apoptosis are well known in the art and may be used or routinely modified to assess the ability of
		_	in immune cells	polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to
			(such as	promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase
	•		eosinophils).	activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides
			•	of the invention (including antibodies and agonists or antagonists of the invention) include the
				assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res
				247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin,
				Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the
				contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that
-				may be used according to these assays include eosinophils. Eosinophils are important in the late
				stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of
				late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to
				assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists
				of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in
				eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in
				dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-
				activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000);
				Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med;
				Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR,
				et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced
				phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal
				kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of
				each of which are herein incorporated by reference in its entirety.
41	HBINS58	999	Production of	TNFa FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells,
			TNF alpha by	fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and

			dendritic cells	cytotoxic effects on a variety of cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or	3 of
	_			entagonists of the invention) to mediate infinitionalisation, inodulate initialisation and experience extensions. Exemplary assays that test for immunomodulatory proteins evaluate the production of	n of
				cytokines such as tumor necrosis factor alpha (11NFa), and the induction of innibilion of an inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test	
				immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or	o
				antagonists of the invention) include assays disclosed in Miragila et al., J Biomolecular Screening 4.103.204(1000): Dowland et al. "I vmmhoovtee: a practical approach" Chapter 6.138-160 (2000):	 e :
				4.153-204(1537), NOWIGHU et al., Editablication, a practical approach. Crapica (2007), Verhasselt et al., Eur J Immunol 28(11):3886-3890 (1198); Dahlen et al., J Immunol 160(7):3585-	
				3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol	3iol
				65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety.	irety.
				Human dendritic cells that may be used according to these assays may be isolated using techniques	nes
				disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in	lls in
				suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell	leo]
				proliferation and functional activities.	
41	HBINS58	999	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely	×
				modified to assess the ability of polypeptides of the invention (including antibodies and agonists or	s or
				antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is	_
				measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is	ls is
				upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component	ent
				in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of	
				insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and	and
				agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J,	·. ·
				47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et	K., et
				al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52	,
				(1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of	nts of
				each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used	sed
				according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely	ار کران
				generated. Exemplary pancreatic cells that may be used according to these assays include HITT15	[15]
				Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells	-
				transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors.	rs.
				The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by	
				somatostatin or glucocorticoids. A11C# CKL-1/// Refs: Lord and Ashcroft. Biochem. J. 219:	<u></u>

				547-551: Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343. 1981.
45	HBJID05		Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease." Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma. Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL 10, IL 13, IL 5 and IL 6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
43	HBJJU28	899	Production of IL- 13 and activation of T-cells.	Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science;282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL.13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
44	HBJNC59	699	Activation of T-Cell p38 or JNK	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the

			Pathway.	invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists of the invention) include the assays disclosed the prore tell. Biol Chem
				379(8-9):1101-1110 (1996); Cupia et al., Exp. Cell Res. 247(2): 493-304 (1999), Fylladds JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein
				incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according
				to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
45	HBMCI50	0/9	Production of IL-8	Assay that measures the production of the chemokine interleukin-8 (IL-8) from immune cells (such
			(such as the	as the EQL-1 human cosmophic centimes are well known in the art (not example, incasurement of IL-8 production by FMAT) and may be used or routinely modified to assess the ability of
			human EOL-1	polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to
			eosinophil cells)	promote or inhibit. Eosinophils are a type of immune cell important in allergic responses; they are
				recruited to itssues and mediate the initial filling response of face stage affected reaction. Let us a
				and disorders (such as allergy and asthma).
45	HBMCI50	0/9	Activation of	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast
			transcription	cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
			through GATA-3	production. Assays for the activation of transcription through the GATA3 response element are
			response element	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
			in immune cells	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
			(such as mast	transcription factors and modulate expression of mast cell genes important for immune response
			cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test CATA3-response element activity of polypeptides of the
				Invention (including annocates and agonists of antagonists of the invention) include assays disclosed in Berger et al. Gene 66-1-10 (1008): Cullen and Malm. Methods in Brzymol 216-362.
				488 (1992): Henthorn et al., Proc Natl Acad Sci 115A 85:6342-6346 (1988): Flavell et al., Cold
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available

(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.		ion of Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to meaure the upregulation of cell surface VCAM-1 expression in endothelial cells. Endothelial cells and include human unabilical vein endothelial cells that may be used according to these assays include human unabilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors,
	ls ls	
	0.00	0.29
	HBMCI50	HBMCIS0
	45	45

45	HBMCI50	0.00	Production of IL-8 by by endothelial cells (such as Human Umbilical Cord Endothelial Cells).	Assays measuring production of IL-8 are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate production and/or secretion of IL-8. For example, FMAT may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate production and/or secretion of IL-8 from endothelial cells (such as human umbilical vein endothelial cells (HUVEC)). HUVECs are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Endothelial cells play a pivotal role in the initiation and perpetuation of inflammation and secretion of IL-8 may play an important role in recruitment and activation of immune cells such as neutrophils, macrophages, and lymphocytes.
45	HBMCI50	670	Production of ICAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Endothelial cells, which are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used in ICAM production assays include human umbilical vein endothelial cells (HUVEC), and are available from commercial sources. The expression of ICAM (CD54), a intergral membrane protein, can be upregulated by cytokines or other factors, and ICAM expression is important in mediating immune and endothelial cell interactions leading to immune and inflammatory responses. Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety.
46	HBNAW17	671	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated

46	HBNAW17	671	Insulin Secretion Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an LL-2 dependent suspension culture of T cells with cytotoxic activity. Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr. J. 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., 18 iol Chem. 271(28): 16544-52 according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoids. ATCK-RCL-1777 Refs. Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981. Kinase assay. DNK kinase assays for signal transduction that regulate cell proliferation,
				assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Notes 107604037
				contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late

84	HCACUS8	673	Activation of	stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety. Assays for the activation of transcription through the Serum Response Element (SRE) are well-
			transcription through serum response element in immune cells (such as T-cells).	known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.
84 8	HCACU58	673	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the

48	HCACU58	673	Production of ICAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC)) Production of IL-10 and activation of T-cells.	invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Bur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 86/4):877-360 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the mast cells that ine blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extrawastion. Exemplary endothelial cells that may be used in ICAM production assays include thuman umbilical vein endothelial cells that may be used in ICAM production assays include expression of ICAM (CD54) a intergral membrane protein, can be upregulated by cytokines or other factors, and ICAM expression is important in mediating immune and endothelial cell interactions leading to immune and inflammatory responses. Assays for measuring expression of ICAM expression is endothelial cells that may be used or routinely modified to measure ICAM-1 are well-known in the art and may be used or routinely modified to measure ICAM-1 are expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Ir, et al., Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety. Assays for production of LL-10 and activation of T-cells are
		_		modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-
				Therapeutics, 88: 187-196 (2000); the contents of each of which are herein incorporated by

49	HCDAF84	674	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood. This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998), Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999), Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
46	HCDAF84	674	Activation of transcription through NFAT response element	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the
			in immune cells (such as mast cells).	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)

				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
49	HCDAF84	674	Production of VCAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to meaure the upregulation of cell surface VCAM-I expression in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors, and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.
49	HCDAF84	674	Production of ICAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Endothelial cells, which are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used in ICAM production assays include human umbilical vein endothelial cells (HUVEC), and are available from commercial sources. The expression of ICAM (CD54), a intergral membrane protein, can be upregulated by cytokines or other factors, and ICAM expression is important in mediating immune and endothelial cell interactions leading to immune and inflammatory responses. Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein

				:
65	HCE2E4	313	Domilation of	A sense for the reministion of transcription through the DEDOV promotes are used bround in the ort
3	177771		transcription	assays for the regulation of transcription through the 1 El Civ promoted are well-known in the arc
			through the	(including antibodies and agonists or antagonists of the invention) to activate the PEPCK promoter
			PEPCK promoter	in a reporter construct and regulate liver gluconeogenesis. Exemplary assays for regulation of
			in hepatocytes	transcription through the PEPCK promoter that may be used or routinely modified to test for
				PEPCK promoter activity (in hepatocytes) of polypeptides of the invention (including antibodies
				and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10
				(1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl
				Acad Sci USA 85:6342-6346 (1988); Lochhead et al., Diabetes 49(6):896-903 (2000); and Yeagley
				et al., J Biol Chem 275(23):17814-17820 (2000), the contents of each of which is herein
				incorporated by reference in its entirety. Hepatocyte cells that may be used according to these
				assays are publicly available (e.g., through the ATCC) and/or may be routinely generated.
				Exemplary liver hepatoma cells that may be used according to these assays include H4lle cells,
				which contain a tyrosine amino transferase that is inducible with glucocorticoids, insulin, or cAMP
				derivatives.
20	HCE2F54	675	Activation of	Assays for the activation of transcription through the NFKB response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through NFKB	(including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription
			response element	factors and modulate expression of epithhelial genes. Exemplary assays for transcription through
			in epithelial cells	the NFKB response element that may be used or routinely modified to test NFKB-response element
			(such as HELA	activity of polypeptides of the invention (including antibodies and agonists or antagonists of the
			cells).	invention) include assays disclosed in: Kaltschmidt B, et al., Oncogene, 18(21):3213-3225 (1999);
				Beetz A, et al., Int J Radiat Biol, 76(11):1443-1453 (2000); Berger et al., Gene 66:1-10 (1998);
				Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci
				USA 85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et
				al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each
				of which are herein incorporated by reference in its entirety. Epithelial cells that may be used
				according to these assays are publicly available (e.g., through the ATCC). Exemplary epithelial
	,			cells that may be used according to these assays include the HELA cell line.
20	HCE2F54	675	Activation of	This assay uses a NFKB response element (which will bind NFKB transcription factors) linked to a
			transcription	reporter gene to measure NFKB mediated transcription in the human monocyte cell line U937.
			through NFKB	NFKB is upregulated by cytokines and other factors and NFKB element activation leads to
			response element	expression of immunomodulatory genes. Activation of NFKB in monocytes can play a role in

in immune cells (such as the U937 human monocyte cell line). Stimulation of insulin secretion from pancreatic beta cells. Production of IL-10 and activation of T-cells.	HCE3G69 676 Stimulation of insulin secretion from pancreatic beta cells. HCE3G69 676 Stimulation of insulin secretion from pancreatic beta cells. HCE3G69 676 Production of II 10 and activatio of T-cells.

and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL 10, IL 13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-704 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology &
	Stimulation of insulin secretion from pancreatic beta cells.	Production of IL- 10 and activation of T-cells.
		678
	HCE5F43	HCEEA88
	52	53

agonists or antagonists of the invention) to stimulate or inhibit production of LL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate LL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "Th-elper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL 10, IL 13, IL 5 and IL 6. Factors that induce differentiation and activation of Th2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary mouse T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the CTLL cell line, which is a suspension culture of IL-2 dependent cytotoxic T cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and
agonists or antagonists of the instruction of T-cells. Exemplary assays toolypeptides and antibodies of modulate IL-10 production and and/or cited in: Robinson, DS, 968 (2000), and Cohn, et al., "Therapeutics; 88: 187-196 (200 reference in their entirety. Exercibles. IL 10 secreted from Th2 are a class of T cells that secretand activation of Th2 cells plansthma. Primary T helper 2 or using peripheral blood lympho	Assays for the activation of transcription through the G response element are well-known in the art and may be ability of polypeptides of the invention (including antibinvention) to regulate STAT transcription factors and m variety of cell functions. Exemplary assays for transcrimay be used or routinely modified to test GAS-respons invention (including antibodies and agonists or antagon disclosed in Berger et al., Gene 66:1-10 (1998); Cullen 368 (1992); Henthorn et al., Proc Natl Acad Sci USA 8 Blood 93(6):1980-1991 (1999); and Henttinen et al., JI contents of each of which are herein incorporated by recells that may be used according to these suspension culture of IL-2 dependent cytotoxic T cells.	Assays for measuring secretion modified to assess the ability of antagonists of the invention) to measured by FMAT using antipupregulated by glucose and also diabetes. Exemplary assays in diabetes.
of T-cells.		Insulin Secretion
	089	089
	нсегв80	HCEFB80
	55	55

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<220>
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His Val Cys Phe Cys Pro Arg Leu Cys Leu Ala Val Pro Cys Val Phe
His Leu Ser Ser Pro Trp Phe His Val Arg Xaa Xaa Phe Phe Ser Gly
Xaa Pro Gly Cys Ile Trp Gly Ile Cys Phe Val Gly Leu Leu Gly
Ala Xaa Arg Pro Arg Ser Gly Cys Leu Cys Ser Pro Ser Xaa Cys Leu
Trp Ser Leu Val Val Cys Glu Ser Ile Cys Leu Pro Arg Xaa Gly Pro
Asn Gln Ala Pro Pro Xaa Pro Leu Phe Leu Ser Leu Asn Leu Pro Phe
Leu Phe Gln Pro Leu Gln Met Arg Trp Leu Ser Ala Val Gly Trp Arg
                            120
Glu Ala Met
    130
<210> 631
<211> 182
<212> PRT
<213> Homo sapiens
<400> 631
Met Lys Gly Trp Gly Trp Leu Ala Leu Leu Gly Ala Leu Leu Gly
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Thr Ala Trp Ala Arg Arg Ser Gln Asp Leu His Cys Gly Ala Cys Arg
20 25 30

Ala Leu Val Asp Glu Leu Glu Trp Glu Ile Ala Gln Val Asp Pro Lys
35 40 45

Ser Val Val Glu Val Pro Tyr Ala Arg Ser Glu Ala His Leu Thr Glu 65 70 75 80

Leu Leu Glu Glu Ile Cys Asp Arg Met Lys Glu Tyr Gly Glu Gln Ile 85 90 95

Asp Pro Ser Thr His Arg Lys Asn Tyr Val Arg Val Val Gly Arg Asn 100 105 110

Gly Glu Ser Ser Glu Leu Asp Leu Gln Gly Ile Arg Ile Asp Ser Asp 115 120 125

Ile Ser Gly Thr Leu Lys Phe Ala Cys Glu Ser Ile Val Glu Glu Tyr 130 135 140

Glu Asp Glu Leu Ile Glu Phe Phe Ser Arg Glu Ala Asp Asn Val Lys 145 150 155 160

Asp Lys Leu Cys Ser Lys Arg Thr Asp Leu Cys Asp His Ala Leu His 165 170 175

Ile Ser His Asp Glu Leu 180

<210> 632

<211> 49

<212> PRT

<213> Homo sapiens

<400> 632

Met Arg Leu Cys Ser Phe Thr Lys Val Pro Met Asn Leu Phe Leu Asn 1 5 10 15

Val Ile Leu Leu Lys Phe Tyr Asn Phe Leu Phe Ser Leu Ile Leu Gly
20 25 30

Lys Ser Cys Leu Ala Ser Leu Gly Leu Cys Lys Asn Asn Lys Cys Leu $35 \hspace{1cm} 40 \hspace{1cm} 45$

Ser

<210> 633

<211> 218

<212> PRT

<213> Homo sapiens

Met Gly Ser Ala Ala Leu Glu Ile Leu Gly Leu Val Leu Cys Leu Val 10 Gly Trp Gly Gly Leu Ile Leu Ala Cys Gly Leu Pro Met Trp Gln Val Thr Ala Phe Leu Asp His Asn Ile Val Thr Ala Gln Thr Thr Trp Lys 40 Gly Leu Trp Met Ser Cys Val Val Gln Ser Thr Gly His Met Gln Cys Lys Val Tyr Asp Ser Val Leu Ala Leu Ser Thr Glu Val Gln Ala Ala Arg Ala Leu Thr Val Ser Ala Val Leu Leu Ala Phe Val Ala Leu Phe Val Thr Leu Ala Gly Ala Gln Cys Thr Thr Cys Val Ala Pro Gly Pro Ala Lys Ala Arg Val Ala Leu Thr Gly Gly Val Leu Tyr Leu Phe Cys Gly Leu Leu Ala Leu Val Pro Leu Cys Trp Phe Ala Asn Ile Val Val 135 Arg Glu Phe Tyr Asp Pro Ser Val Pro Val Ser Gln Lys Tyr Glu Leu Gly Ala Ala Leu Tyr Ile Gly Trp Ala Ala Thr Ala Leu Leu Met Val Gly Gly Cys Leu Leu Cys Cys Gly Ala Trp Val Cys Thr Gly Arg Pro Asp Leu Ser Phe Pro Val Lys Tyr Ser Ala Pro Arg Arg Pro Thr Ala 200 Thr Gly Asp Tyr Asp Lys Lys Asn Tyr Val

<210> 634 <211> 30 <212> PRT <213> Homo sapiens

<400> 634
Met Ala Leu Ser Val Leu Val Leu Leu Leu Leu Ala Val Leu Tyr Glu
1 5 10 15

Gly Ile Lys Val Gly Lys Ala Ser Cys Ser Thr Arg Tyr Trp 20 25 30

<210> 635 <211> 62

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<212> PRT
<213> Homo sapiens
<220>
<221> SITE
<222> (16)
<223> Xaa equals any amino acid
<220>
<221> SITE
<222> (54)
<223> Xaa equals any amino acid
<400> 635
Met Val Thr Gly Phe Phe Phe Ile Leu Met Thr Val Leu Trp Phe Xaa
Arg Glu Pro Gly Phe Val Pro Gly Trp Asp Ser Phe Phe Glu Lys Lys
                                 25
Gly Tyr Arg Thr Asp Ala Thr Val Ser Val Phe Leu Gly Phe Leu Leu
Phe Leu Ile Pro Ala Xaa Glu Ala Leu Leu Trp Glu Lys Glu
                        55
<210> 636
<211> 93
<212> PRT
<213> Homo sapiens
<400> 636
Met Pro Arg Ala Thr Leu Trp Gly His Leu Ser Pro Ala Trp Val Leu
Val Pro Trp Thr Pro Arg Ala Cys Gly Gln Ala Ala Pro Gly Arg Gly
His Val Ala Ser Asp His Lys Ser Gly Leu Pro Trp Pro Lys His Cys
Ser Cys Leu His Pro Arg Ala Ser Gln Pro Cys Leu Phe Ser Leu Asn
Ser Asn Arg Thr Val Phe Thr Ala Ile Gln Arg Val Ala Leu Gly Trp
                                         75
Thr Phe Trp Val Gln Ala Asn Leu Val Pro Arg Cys Thr
                 85
<210> 637
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<211> 122 <212> PRT <213> Homo sapiens

<400> 637

Met Cys Tyr Leu Leu Leu Leu Ile Gln Thr Ala Glu Leu Leu Ile

10 His Pro Gln Gly Leu Gln Ala Val Ser Asn Gly Glu Ser Ala Leu Lys 25 20 Gly Thr Arg Pro Thr Phe Ser Ser Pro Phe Ile Leu Val Thr Glu Gly Arg Lys Glu Trp Glu Gly Val Phe Leu Ser Ser Gly Trp Lys Gly Asn Thr Leu Ser Asn Tyr Tyr Ile Ser Leu Val Phe Tyr Tyr Ser Arg Ile Leu Gln Pro Tyr Phe Tyr Cys Leu Trp Gly Lys Leu Glu Met Val Thr Leu Ile Arg Ser Val Trp Arg Gly Ile Asn Gly Gly Asp Lys Ile Gln Leu Val Leu Glu Asn Val Lys Val Leu Lys <210> 638 <211> 198 <212> PRT <213> Homo sapiens <220> <221> SITE <222> (29) <223> Xaa equals any amino acid <400> 638 Met Lys Lys Ser Leu Glu Asn Leu Asn Arg Leu Gln Val Met Leu Leu 10 His Leu Thr Ala Ala Phe Leu Gln Arg Ala Gln His Xaa Phe Asp Tyr Lys Asp Glu Ser Gly Phe Pro Lys Pro Pro Ser Tyr Asn Val Ala Thr Thr Leu Pro Ser Tyr Asp Glu Ala Glu Arg Thr Lys Ala Glu Ala Thr Ile Pro Leu Val Pro Gly Arg Asp Glu Asp Phe Val Gly Arg Asp Asp Phe Asp Asp Ala Asp Gln Leu Arg Ile Gly Asn Asp Gly Ile Phe Met Leu Thr Phe Phe Met Ala Phe Leu Phe Asn Trp Ile Gly Phe Phe Leu Ser Phe Cys Leu Thr Thr Ser Ala Ala Gly Arg Tyr Gly Ala Ile Ser 120

Gly Phe Gly Leu Ser Leu Ile Lys Trp Ile Leu Ile Val Arg Phe Ser

135 130 Thr Tyr Phe Pro Gly Tyr Phe Asp Gly Gln Tyr Trp Leu Trp Trp Val 150 Phe Leu Val Leu Gly Phe Leu Leu Phe Leu Arg Gly Phe Ile Asn Tyr Ala Lys Val Arg Lys Met Pro Glu Thr Phe Ser Asn Leu Pro Arg Thr 185 Arg Val Leu Phe Ile Tyr 195 <210> 639 <211> 66 <212> PRT <213> Homo sapiens <220> <221> SITE <222> (12) <223> Xaa equals any amino acid <400> 639 Met Asn Leu Ser Ile Ile Leu Pro Asn Ser Phe Xaa His Leu Cys Asn 10 Phe Ser Leu Phe Leu Leu Pro Leu Pro Val Pro Ser Gln Pro Leu Ile Cys Ser Gly Asn Tyr Gln Ser Ser Phe Cys His Tyr Arg Leu Ile Cys Ile Phe Lys Glu Ile Tyr Ile His Gly Thr Ile His His Leu Cys Phe Val Val 65 <210> 640 <211> 317 <212> PRT <213> Homo sapiens <220> <221> SITE <222> (207) <223> Xaa equals any amino acid Met Pro Gly Leu Gly Arg Pro Arg Gln Ala Arg Trp Thr Leu Met Leu Leu Leu Ser Thr Ala Met Tyr Gly Ala His Ala Pro Leu Leu Ala Leu

25

Cys His Val Asp Gly Arg Val Pro Phe Arg Pro Ser Ser Ala Val Leu $35 \hspace{1cm} 40 \hspace{1cm} 45$

- Leu Thr Glu Leu Thr Lys Leu Leu Leu Cys Ala Phe Ser Leu Leu Val 50 55 60
- Gly Trp Gln Ala Trp Pro Gln Gly Pro Pro Pro Trp Arg Gln Ala Ala 65 70 75 80
- Pro Phe Ala Leu Ser Ala Leu Leu Tyr Gly Ala Asn Asn Asn Leu Val 85 90 95
- Ile Tyr Leu Gln Arg Tyr Met Asp Pro Ser Thr Tyr Gln Val Leu Ser 100 105 110
- Asn Leu Lys Ile Gly Ser Thr Ala Val Leu Tyr Cys Leu Cys Leu Arg 115 120 125
- His Arg Leu Ser Val Arg Gln Gly Leu Ala Leu Leu Leu Leu Met Ala 130 135 140
- Ala Gly Ala Cys Tyr Ala Ala Gly Gly Leu Gln Val Pro Gly Asn Thr 145 150 155 160
- Leu Pro Ser Pro Pro Pro Ala Ala Ala Ser Pro Met Pro Leu His 165 170 175
- Ile Thr Pro Leu Gly Leu Leu Leu Leu Ile Leu Tyr Cys Leu Ile Ser 180 185 190
- Gly Leu Ser Ser Val Tyr Thr Glu Leu Leu Met Lys Arg Gln Xaa Leu 195 200 205
- Pro Leu Ala Leu Gln Asn Leu Phe Leu Tyr Thr Phe Gly Val Leu Leu 210 215 220
- Asn Leu Gly Leu His Ala Gly Gly Gly Ser Gly Pro Gly Leu Leu Glu 225 230 235 240
- Gly Phe Ser Gly Trp Ala Ala Leu Val Val Leu Ser Gln Ala Leu Asn 245 250 255
- Gly Leu Leu Met Ser Ala Val Met Lys His Gly Ser Ser Ile Thr Arg 260 265 270
- Leu Phe Val Val Ser Cys Ser Leu Val Val Asn Ala Val Leu Ser Ala 275 280 285
- Val Leu Leu Arg Leu Gln Leu Thr Ala Ala Phe Phe Leu Ala Thr Leu 290 295 300
- Leu Ile Gly Leu Ala Met Arg Leu Tyr Tyr Gly Ser Arg 305 310 315

<210> 641

<211> 446

<212> PRT

<213> Homo sapiens

)> 64										_		_		
Met 1	Leu	Leu	Gly	Leu 5	Leu	Met	Ala	Ala	Cys 10	Phe	Thr	Phe	Cys	Leu 15	Ser
His	Gln	Asn	Leu 20	Lys	Glu	Phe	Ala	Leu 25	Thr	Asn	Pro	Glu	J 30	Ser	Ser
Thr	Lys	Glu 35	Thr	Glu	Arg	Lys	Glu 40	Thr	Lys	Ala	Glu	Glu 45	Glu	Leu	Asp
Ala	Glu 50	Val	Leu	Glu	Val	Phe 55	His	Pro	Thr	His	Glu 60	Trp	Gln	Ala	Leu
Gln 65	Pro	Gly	Gln	Ala	Val 70	Pro	Ala	Gly	Ser	His 75	Val	Arg	Leu	Asn	Leu 80
Gln	Thr	Gly	Glu	Arg 85	Glu	Ala	Lys	Leu	Gln 90	Tyr	Glu	Asp	Lys	Phe 95	Arg
Asn	Asn	Leu	Lys 100	Gly	Lys	Arg	Leu	Asp 105	Ile	Asn	Thr	Asn	Thr 110	Tyr	Thr
Ser	Gln	Asp 115	Leu	Lys	Ser	Ala	Leu 120	Ala	Lys	Phe	Lys	Glu 125	Gly	Ala	Glu
Met	Glu 130	Ser	Ser	Lys	Glu	Asp 135	Lys	Ala	Arg	Gln	Ala 140	Glu	Val	Lys	Arg
Leu 145	Phe	Arg	Pro	Ile	Glu 150	Glu	Leu	Lys	Lys	Asp 155	Phe	Asp	Glu	Leu	Asn 160
Val	Val	Ile	Glu	Thr 165	Asp	Met	Gln	Ile	Met 170	Val	Arg	Leu	Ile	Asn 175	Lys
Phe	Asn	Ser	Ser 180	Ser	Ser	Ser	Leu	Glu 185	Glu	Lys	Ile	Ala	Ala 190	Leu	Phe
Asp	Leu	Glu 195	Tyr	Tyr	Val	His	Gln 200	Met	Asp	Asn	Ala	Gln 205	Asp	Leu	Leu
Ser	Phe 210	Gly	Gly	Leu	Gln	Val 215	Val	Ile	Asn	Gly	Leu 220	Asn	Ser	Thr	Glu
Pro 225	Leu	Val	Lys	Glu	Tyr 230	Ala	Ala	Phe	Val	Leu 235	Gly	Ala	Ala	Phe	Ser 240
Ser	Asn	Pro	Lys	Val 245	Gln	Val	Glu	Ala	Ile 250	Glu	Gly	Gly	Ala	Leu 255	Gln
Lys	Leu	Leu	Val 260	Ile	Leu	Ala	Thr	Glu 265	Gln	Pro	Leu	Thr	Ala 270	Lys	Lys
Lys	Val	Leu 275	Phe	Ala	Leu	Суѕ	Ser 280	Leu	Leu	Arg	His	Phe 285	Pro	Tyr	Ala
Gln	Arg 290	Gln	Phe	Leu	Lys	Leu 295	Gly	Gly	Leu	Gln	Val 300	Leu	Arg	Thr	Leu

Val Gln Glu Lys Gly Thr Glu Val Leu Ala Val Arg Val Val Thr Leu

Leu Tyr Asp Leu Val Thr Glu Lys Met Phe Ala Glu Glu Glu Ala Glu 325 330 335

Leu Thr Gln Glu Met Ser Pro Glu Lys Leu Gln Gln Tyr Arg Gln Val 340 345 350

His Leu Leu Pro Gly Leu Trp Glu Gln Gly Trp Cys Glu Ile Thr Ala 355 360 365

His Leu Leu Ala Leu Pro Glu His Asp Ala Arg Glu Lys Val Leu Gln 370 375 380

Thr Leu Gly Val Leu Leu Thr Thr Cys Arg Asp Arg Tyr Arg Gln Asp 385 390 395 400

Pro Gln Leu Gly Arg Thr Leu Ala Ser Leu Gln Ala Glu Tyr Gln Val 405 410 415

Leu Ala Ser Leu Glu Leu Gln Asp Gly Glu Asp Glu Gly Tyr Phe Gln 420 425 430

Glu Leu Leu Gly Ser Val Asn Ser Leu Leu Lys Glu Leu Arg 435 440 445

<210> 642

<211> 563

<212> PRT

<213> Homo sapiens

<400> 642

Met Trp Ala Val Leu Arg Leu Ala Leu Arg Pro Cys Ala Arg Ala Ser 1 5 10 15

Pro Ala Gly Pro Arg Ala Tyr His Gly Asp Ser Val Ala Ser Leu Gly 20 25 30

Thr Gln Pro Asp Leu Gly Ser Ala Leu Tyr Gln Glu Asn Tyr Lys Gln 35 40

Met Lys Ala Leu Val Asn Gln Leu His Glu Arg Val Glu His Ile Lys 50 55 60

Leu Gly Gly Glu Lys Ala Arg Ala Leu His Ile Ser Arg Gly Lys 65 70 75 80

Leu Leu Pro Arg Glu Arg Ile Asp Asn Leu Ile Asp Pro Gly Ser Pro
85 90 95

Phe Leu Glu Leu Ser Gln Phe Ala Gly Tyr Gln Leu Tyr Asp Asn Glu 100 105 110

Glu Val Pro Gly Gly Gly Ile Ile Thr Gly Ile Gly Arg Val Ser Gly
115 120 125

Val Glu Cys Met Ile Ile Ala Asn Asp Ala Thr Val Lys Gly Gly Ala 130 135 140

Tyr Tyr Pro Val Thr Val Lys Lys Gln Leu Arg Ala Gln Glu Ile Ala 145 150 155 160

Met Gln Asn Arg Leu Pro Cys Ile Tyr Leu Val Asp Ser Gly Gly Ala Tyr Leu Pro Arg Gln Ala Asp Val Phe Pro Asp Arg Asp His Phe Gly 185 Arg Thr Phe Tyr Asn Gln Ala Ile Met Ser Ser Lys Asn Ile Ala Gln 200 Ile Ala Val Val Met Gly Ser Cys Thr Ala Gly Gly Ala Tyr Val Pro 215 Ala Met Ala Asp Glu Asn Ile Ile Val Arg Lys Gln Gly Thr Ile Phe Leu Ala Gly Pro Pro Leu Val Lys Ala Ala Thr Gly Glu Glu Val Ser Ala Glu Asp Leu Gly Gly Ala Asp Leu His Cys Arg Lys Ser Gly Val Ser Asp His Trp Ala Leu Asp Asp His His Ala Leu His Leu Thr Arg 280 Lys Val Val Arg Asn Leu Asn Tyr Gln Lys Lys Leu Asp Val Thr Ile Glu Pro Ser Glu Glu Pro Leu Phe Pro Ala Asp Glu Leu Tyr Gly Ile 315 Val Gly Ala Asn Leu Lys Arg Ser Phe Asp Val Arg Glu Val Ile Ala Arg Ile Val Asp Gly Ser Arg Phe Thr Glu Phe Lys Ala Phe Tyr Gly 345 Asp Thr Leu Val Thr Gly Phe Ala Arg Ile Phe Gly Tyr Pro Val Gly 360 Ile Val Gly Asn Asn Gly Val Leu Phe Ser Glu Ser Ala Lys Lys Gly Thr His Phe Val Gln Leu Cys Cys Gln Arg Asn Ile Pro Leu Leu Phe 395 Leu Gln Asn Ile Thr Gly Phe Met Val Gly Arg Glu Tyr Glu Ala Glu Gly Ile Ala Lys Asp Gly Ala Lys Met Val Ala Ala Val Ala Cys Ala 425 Gln Val Pro Lys Ile Thr Leu Ile Ile Gly Gly Ser Tyr Gly Ala Gly 440 Asn Tyr Gly Met Cys Gly Arg Ala Tyr Ser Pro Arg Phe Leu Tyr Ile 460 Trp Pro Asn Ala Arg Ile Ser Val Met Gly Glu Glu Ala Ala Asn 475 470

Val Leu Ala Thr Ile Thr Lys Asp Gln Arg Ala Arg Glu Gly Lys Gln
485 490 495

Phe Ser Ser Ala Asp Glu Ala Ala Leu Lys Glu Pro Ile Ile Lys Lys 500 505 510

Phe Glu Glu Gly Asn Pro Tyr Tyr Ser Ser Ala Arg Val Trp Asp 515 520 525

Asp Gly Ile Ile Asp Pro Ala Asp Thr Arg Leu Val Leu Gly Leu Ser 530 535 540

Phe Ser Ala Ala Leu Asn Ala Pro Ile Glu Lys Thr Asp Phe Gly Ile 545 550 560

Phe Arg Met

<210> 643

<211> 53

<212> PRT

<213> Homo sapiens

<400> 643

Met Val Gln Phe Glu Val Ile Phe Leu Leu Phe Gly Leu Cys Phe Ser 1 5 10 15

Ser Ser Ser Ser Arg Leu Val Gly Ser Gln Val Glu Asn Phe Ser Pro

Thr Pro Cys Ile Phe Gln Ala Phe Arg Cys Ser Ser Leu Ala Ile Ile 35 40 45

Ser Met Ser Leu Ser 50

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<211> 607

<212> PRT

<213> Homo sapiens

<220>

<221> SITE

<222> (242)

<223> Xaa equals any amino acid

<400> 644

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Pro Asp Gly Val Arg Pro Gln Pro Ser Ser Ser Pro Ser Gly Ala Val 20 25 30

Pro Thr Ser Leu Glu Leu Gln Arg Gly Thr Asp Gly Gly Thr Leu Gln

Ser Pro Ser Glu Ala Thr Ala Thr Arg Pro Ala Val Pro Gly Leu Pro

	50					55					60				
Thr 65	Val	Val	Pro	Thr	Leu 70	Val	Thr	Pro	Ser	Ala 75	Pro	Gly	Asn	Arg	Thr 80
Val	Asp	Leu	Phe	Pro 85	Val	Leu	Pro	Ile	Суs 90	Val	Суѕ	Asp	Leu	Thr 95	Pro
Gly	Ala	Суѕ	Asp 100	Ile	Asn	Суз	Cys	Cys 105	Asp	Arg	Asp	Cys	Tyr 110	Leu	Leu
His	Pro	Arg 115	Thr	Val	Phe	Ser	Phe 120	Суѕ	Leu	Pro	Gly	Ser 125	Val	Arg	Ser
Ser	Ser 130	Trp	Val	Cys	Val	Asp 135	Asn	Ser	Val	Ile	Phe 140	Arg	Ser	Asn	Ser
Pro 145	Phe	Pro	Ser	Arg	Val 150	Phe	Met	Asp	Ser	Asn 155	Gly	Ile	Arg	Gln	Phe 160
_				165					170					Leu 175	
			180					185					190	Gly	
		195					200					205		Phe	
	210					215					220			Val	
225					230					235				Cys	240
				245					250					Cys 255	
			260					265					270	Ala	
		275					280					285		Arg	
	290					295					300			Leu	
305					310					315				Asn	320
				325					330					Gly 335	
			340					345					350	Glu	
		355					360					365		Phe	
Gln	Ser 370		Ala	Ala	Ser	Leu 375	Thr	Ser	Pro	Arg	Ser 380	Gly	Asn	Pro	Gly

Tyr Ile Val Gly Lys Pro Leu Leu Ala Leu Thr Asp Asp Ile Ser Tyr 395 Ser Met Thr Leu Leu Gln Ser Gln Gly Asn Gly Ser Cys Ser Val Lys 405 Arg His Glu Val Gln Phe Gly Val Asn Ala Ile Ser Gly Cys Lys Leu Arg Leu Lys Lys Ala Asp Cys Ser His Leu Gln Gln Glu Ile Tyr Gln 440 Thr Leu His Gly Arg Pro Arg Pro Glu Tyr Val Ala Ile Phe Gly Asn 455 Ala Asp Pro Ala Gln Lys Gly Gly Trp Thr Arg Ile Leu Asn Arg His Cys Ser Ile Ser Ala Ile Asn Cys Thr Ser Cys Cys Leu Ile Pro Val Ser Leu Glu Ile Gln Val Leu Trp Ala Tyr Val Gly Leu Leu Ser Asn 505 Pro Gln Ala His Val Ser Gly Val Arg Phe Leu Tyr Gln Cys Gln Ser 520 Ile Gln Asp Ser Gln Gln Val Thr Glu Val Ser Leu Thr Thr Leu Val Asn Phe Val Asp Ile Thr Gln Lys Pro Gln Pro Pro Arg Gly Gln Pro Lys Met Asp Trp Lys Trp Pro Phe Asp Phe Phe Pro Phe Lys Val Ala 570 Phe Ser Arg Gly Val Phe Ser Gln Lys Cys Ser Val Ser Pro Ile Leu Ile Leu Cys Leu Leu Leu Gly Val Leu Asn Leu Glu Thr Met 600

<210> 645 <211> 191 <212> PRT

<213> Homo sapiens

<400> 645

Met Ala Ala Pro Arg Gly Arg Ala Ala Pro Trp Thr Thr Ala Leu Leu 1 5 10 15

Leu Leu Leu Ala Ser Gl
n Val Leu Ser Pro Gly Ser Cys Ala Asp Glu 20 25 30

Glu Glu Val Pro Glu Glu Trp Val Leu His Val Val Gln Gly Gln 35 40 45

Ile Gly Ala Gly Asn Tyr Ser Tyr Leu Arg Leu Asn His Glu Gly Lys

50 55 60

Ile Val Leu Arg Met Arg Ser Leu Lys Gly Asp Ala Asp Leu Tyr Val 65 70 75 80

Ser Ala Ser Ser Leu His Pro Ser Phe Asp Asp Tyr Glu Leu Gln Ser 85 90 95

Ala Thr Cys Gly Pro Asp Ala Val Ser Ile Pro Ala His Phe Arg Arg 100 105 110

Pro Val Gly Ile Gly Val Tyr Gly His Pro Ser His Leu Glu Ser Glu 115 120 125

Phe Glu Met Lys Val Tyr Tyr Asp Gly Thr Val Glu Gln His Pro Phe 130 135 140

Gly Glu Ala Ala Tyr Pro Ala Asp Gly Ala Asp Ala Gly Gln Lys His 145 150 155 160

Ala Gly Ala Pro Glu Asp Ala Ser Gln Glu Glu Glu Ser Val Leu Trp 165 170 175

Thr Ile Leu Ile Ser Ile Leu Lys Leu Glu Leu Glu Ile Leu Phe 180 185 190

<210> 646

<211> 421

<212> PRT

<213> Homo sapiens

<400> 646

Met Thr Val Phe Phe Lys Thr Leu Arg Asn His Trp Lys Lys Thr Thr 1 5 10 15

Ala Gly Leu Cys Leu Leu Thr Trp Gly Gly His Trp Leu Tyr Gly Lys

His Cys Asp Asn Leu Leu Arg Arg Ala Ala Cys Gln Glu Ala Gln Val\$35\$ 40 45

Phe Gly Asn Gln Leu Ile Pro Pro Asn Ala Gln Val Lys Lys Ala Thr 50 55 60

Val Phe Ser Ile Leu Gln Leu Ala Lys Glu Lys Pro Gly Leu Tyr Leu 65 70 75 80

Lys Lys Met Leu Pro Asp Phe Thr Phe Ile Trp His Gly Cys Asp Tyr 85 90 95

Cys Lys Thr Asp Tyr Glu Gly Gln Ala Lys Lys Leu Leu Glu Leu Met 100 105 110

Glu Asn Thr Asp Val Ile Ile Val Ala Gly Gly Asp Gly Thr Leu Gln 115 120 125

Glu Val Val Thr Gly Val Leu Arg Arg Thr Asp Glu Ala Thr Phe Ser 130 135 140

Lys Ile Pro Ile Gly Phe Ile Pro Leu Gly Glu Thr Ser Ser Leu Ser 145 150 155 160

His Thr Leu Phe Ala Glu Ser Gly Asn Lys Val Gln His Ile Thr Asp 165 170 175

Ala Thr Leu Ala Ile Val Lys Gly Glu Thr Val Pro Leu Asp Val Leu 180 185 190

Gln Ile Lys Gly Glu Lys Glu Gln Pro Val Phe Ala Met Thr Gly Leu 195 200 205

Arg Trp Gly Ser Phe Arg Asp Ala Gly Val Lys Val Ser Lys Tyr Trp 210 215 220

Tyr Leu Gly Pro Leu Lys Ile Lys Ala Ala His Phe Phe Ser Thr Leu 225 230 235 240

Lys Glu Trp Pro Gln Thr His Gln Ala Ser Ile Ser Tyr Thr Gly Pro $245 \hspace{1cm} 250 \hspace{1cm} 255 \hspace{1cm}$

Thr Glu Arg Pro Pro Asn Glu Pro Glu Glu Thr Pro Val Gln Arg Pro 260 265 270

Ser Leu Tyr Arg Arg Ile Leu Arg Arg Leu Ala Ser Tyr Trp Ala Gln 275 280 285

Pro Gln Asp Ala Leu Ser Gln Glu Val Ser Pro Glu Val Trp Lys Asp 290 295 300

Val Gln Leu Ser Thr Ile Glu Leu Ser Ile Thr Thr Arg Asn Asn Gln 305 310 315 320

Leu Asp Pro Thr Ser Lys Glu Asp Phe Leu Asn Ile Cys Ile Glu Pro 325 330 335

Asp Thr Ile Ser Lys Gly Asp Phe Ile Thr Ile Gly Ser Arg Lys Val 340 345 350

Arg Asn Pro Lys Leu His Val Glu Gly Thr Glu Cys Leu Gln Ala Ser 355 360 365

Gln Cys Thr Leu Leu Ile Pro Glu Gly Ala Gly Gly Ser Phe Ser Ile 370 375 380

Asp Ser Glu Glu Tyr Glu Ala Met Pro Val Glu Val Lys Leu Leu Pro 385 390 395 400

Arg Lys Leu Gln Phe Phe Cys Asp Pro Arg Lys Arg Glu Gln Met Leu 405 410 415

Thr Ser Pro Thr Gln 420

<210> 647

<211> 79

<212> PRT

<213> Homo sapiens

<400> 647

Met Asn Tyr Ser Arg Ser Pro Trp Ala Ala Val Met Glu Pro Leu Thr 1 5 10 15

Leu Leu Phe Leu His Leu Ser Cys Leu Leu Ser Leu Cys Glu Ala Val 20 25 30

Gly Trp Asp Ser Glu Cys Leu Val Cys Ser Leu Gly Glu Glu Glu Phe $35 \hspace{1cm} 40 \hspace{1cm} 45$

Leu Arg Met Gln Ala Leu Leu Cys Gly Cys Arg Leu His Leu Gly Gly 50 55 60

Val Leu Tyr Val Cys Thr Leu Gly Thr Ala Cys Ile Trp Lys Ile 65 70 75

<210> 648

<211> 242

<212> PRT

<213> Homo sapiens

<400> 648

Met Gln Leu Gly Ser Val Leu Leu Thr Arg Cys Pro Phe Trp Gly Cys
1 5 10 15

Phe Ser Gln Leu Met Leu Tyr Ala Glu Arg Ala Glu Ala Arg Arg Lys 20 25 30

Pro Asp Ile Pro Val Pro Tyr Leu Tyr Phe Asp Met Gly Ala Ala Val 35 40 45

Leu Cys Ala Ser Phe Met Ser Phe Gly Val Lys Arg Arg Trp Phe Ala 50 55 60

Leu Gly Ala Ala Leu Gln Leu Ala Ile Ser Thr Tyr Ala Ala Tyr Ile 65 70 75 80

Gly Gly Tyr Val His Tyr Gly Asp Trp Leu Lys Val Arg Met Tyr Ser 90 95

Arg Thr Val Ala Ile Ile Gly Gly Phe Leu Val Leu Ala Ser Gly Ala 100 105 110

Gly Glu Leu Tyr Arg Arg Lys Pro Arg Ser Arg Ser Leu Gln Ser Thr 115 120 125

Gly Gln Val Phe Leu Gly Ile Tyr Leu Ile Cys Val Ala Tyr Ser Leu 130 135 140

Gln His Ser Lys Glu Asp Arg Leu Ala Tyr Leu Asn His Leu Pro Gly 145 150 155 160

Gly Glu Leu Met Ile Gln Leu Phe Phe Val Leu Tyr Gly Ile Leu Ala 165 170 175

Leu Ala Phe Leu Ser Gly Tyr Tyr Val Thr Leu Ala Ala Gln Ile Leu 180 185 190

Ala Val Leu Leu Pro Pro Val Met Leu Leu Ile Asp Gly Asn Val Ala

WO 03/038063 PCT/US02/08277 ·

195 200 205

Tyr Trp His Asn Thr Arg Arg Val Glu Phe Trp Asn Gln Met Lys Leu 210 215 220

Leu Gly Glu Ser Val Gly Ile Phe Gly Thr Ala Val Ile Leu Ala Thr 225 230 235 240

Asp Gly

<210> 649

<211> 52

<212> PRT

<213> Homo sapiens

<400> 649

Met Asp Ser Cys Leu Phe Leu Arg Asp Phe Cys Trp Lys Met Arg Met 1 5 10 15

Leu Thr Ile Leu Pro Leu Gly Thr Leu Phe Pro Leu Leu Thr Leu Leu 20 25 30

Leu Leu Pro Leu Glu Val Pro Ser Val Ser Cys Gly Val Pro Phe Ala 35 40 45

Val Trp Asp Leu 50

<210> 650

<211> 189

<212> PRT

<213> Homo sapiens

<220>

<221> SITE

<222> (94)

<223> Xaa equals any amino acid

<400> 650

Met Ala Leu Leu Ser Arg Pro Ala Leu Thr Leu Leu Leu Leu Met
1 5 10 15

Ala Ala Val Val Arg Cys Gln Glu Gln Ala Gln Thr Thr Asp Trp Arg 20 25 30

Ala Thr Leu Lys Thr Ile Arg Asn Gly Val His Lys Ile Asp Thr Tyr 35 40 45

Leu Asn Ala Ala Leu Asp Leu Leu Gly Gly Glu Asp Gly Leu Cys Gln
50 55 60

Tyr Lys Cys Ser Asp Gly Ser Lys Pro Phe Pro Arg Tyr Gly Tyr Lys 65 70 75 80

Pro Ser Pro Pro Asn Gly Cys Gly Ser Pro Leu Phe Gly Xaa His Leu 85 90 95

Asn Ile Gly Ile Pro Ser Leu Thr Lys Cys Cys Asn Gln His Asp Arg 100 105 110

Cys Tyr Glu Thr Cys Gly Lys Ser Lys Asn Asp Cys Asp Glu Glu Phe 115 120 125

Gln Tyr Cys Leu Ser Lys Ile Cys Arg Asp Val Gln Lys Thr Leu Gly 130 135 140

Leu Thr Gln His Val Gln Ala Cys Glu Thr Thr Val Glu Leu Leu Phe 145 150 155 160

Asp Ser Val Ile His Leu Gly Cys Lys Pro Tyr Leu Asp Ser Gln Arg 165 170 175

Ala Ala Cys Arg Cys His Tyr Glu Glu Lys Thr Asp Leu 180 185

<210> 651

<211> 264

<212> PRT

<213> Homo sapiens

<400> 651

Met Leu Arg Cys Gly Gly Arg Gly Leu Leu Cly Leu Ala Val Ala 1 5 10 15

Ala Ala Ala Val Met Ala Ala Arg Leu Met Gly Trp Trp Gly Pro Arg
20 25 30

Ala Gly Phe Arg Leu Phe Ile Pro Glu Glu Leu Ser Arg Tyr Arg Gly
35 40 45

Gly Pro Gly Asp Pro Gly Leu Tyr Leu Ala Leu Leu Gly Arg Val Tyr 50 55 60

Asp Val Ser Ser Gly Arg Arg His Tyr Glu Pro Gly Ser His Tyr Ser 65 70 75 80

Gly Phe Ala Gly Arg Asp Ala Ser Arg Ala Phe Val Thr Gly Asp Cys 85 90 95

Ser Glu Ala Gly Leu Val Asp Asp Val Ser Asp Leu Ser Ala Ala Glu 100 105 110

Met Leu Thr Leu His Asn Trp Leu Ser Phe Tyr Glu Lys Asn Tyr Val 115 120 125

Cys Val Gly Arg Val Thr Gly Arg Phe Tyr Gly Glu Asp Gly Leu Pro 130 135 140

Thr Pro Ala Leu Thr Gln Val Glu Ala Ala Ile Thr Arg Gly Leu Glu 145 150 155 160

Ala Asn Lys Leu Gln Leu Gln Glu Lys Gln Thr Phe Pro Pro Cys Asn 165 170 175

Ala Glu Trp Ser Ser Ala Arg Gly Ser Arg Leu Trp Cys Ser Gln Lys

180 185 190

Ser Gly Gly Val Ser Arg Asp Trp Ile Gly Val Pro Arg Lys Leu Tyr 195 200 205

Lys Pro Gly Ala Lys Glu Pro Arg Cys Val Cys Val Arg Thr Thr Gly 210 215 220

Pro Pro Ser Gly Gln Met Pro Asp Asn Pro Pro His Arg Asn Arg Gly 225 230 235 240

Asp Leu Asp His Pro Asn Leu Ala Glu Tyr Thr Gly Cys Pro Pro Leu 245 250 255

Ala Ile Thr Cys Ser Phe Pro Leu 260

<210> 652

<211> 140

<212> PRT

<213> Homo sapiens

<400> 652

Met Leu Gly Thr Ser Leu Ile Tyr Trp Thr Leu Phe Thr Leu Gly Leu
1 5 10 15

Asp Leu Ser Trp Ser Ile Ser Leu Ala Phe Lys Trp Cys Glu Arg Pro \$20\$

Glu Trp Ile His Val Asp Ser Arg Pro Phe Ala Ser Leu Ser Arg Asp 35 40 45

Ser Gly Ala Ala Leu Gly Leu Gly Ile Ala Leu His Ser Pro Cys Tyr 50 60

Ala Gln Val Arg Arg Ala Gln Leu Gly Asn Gly Gln Lys Ile Ala Cys 65 70 75 80

Leu Val Leu Ala Met Gly Leu Leu Gly Pro Leu Asp Trp Leu Gly His
85 90 95

Pro Pro Gln Ile Ser Leu Phe Tyr Ile Phe Asn Phe Leu Lys Tyr Thr 100 105 110

Leu Trp Pro Cys Leu Val Leu Ala Leu Val Pro Trp Ala Val His Met 115 120 125

Phe Ser Ala Gln Glu Ala Pro Pro Ile His Ser Ser 130 135 140

<210> 653

<211> 248

<212> PRT

<213> Homo sapiens

<400> 653

Met Gly Pro Val Arg Leu Gly Ile Leu Leu Phe Leu Phe Leu Ala Val His Glu Ala Trp Ala Gly Met Leu Lys Glu Glu Asp Asp Asp Thr Glu Arg Leu Pro Ser Lys Cys Glu Val Cys Lys Leu Leu Ser Thr Glu Leu Gln Ala Glu Leu Ser Arg Thr Gly Arg Ser Arg Glu Val Leu Glu Leu Gly Gln Val Leu Asp Thr Gly Lys Arg Lys Arg His Val Pro Tyr Ser Val Ser Glu Thr Arg Leu Glu Glu Ala Leu Glu Asn Leu Cys Glu Arg Ile Leu Asp Tyr Ser Val His Ala Glu Arg Lys Gly Ser Leu Arg Tyr Ala Lys Gly Gln Ser Gln Thr Met Ala Thr Leu Lys Gly Leu Val Gln Lys Gly Val Lys Val Asp Leu Gly Ile Pro Leu Glu Leu Trp Asp Glu 135 Pro Ser Val Glu Val Thr Tyr Leu Lys Lys Gln Cys Glu Thr Met Leu Glu Glu Phe Glu Asp Ile Val Gly Asp Trp Tyr Phe His His Gln Glu 170 Gln Pro Leu Gln Asn Phe Leu Cys Glu Gly His Val Leu Pro Ala Ala Glu Thr Ala Cys Leu Gln Glu Thr Trp Thr Gly Lys Glu Ile Thr Asp 200 Gly Glu Glu Lys Thr Glu Gly Glu Glu Glu Glu Glu Glu Glu Glu Glu Glu Glu Glu Glu Gly Gly Asp Lys Met Thr Lys Thr Gly Ser His

Pro Lys Leu Asp Arg Glu Asp Leu 245

<210> 654 <211> 64 <212> PRT <213> Homo sapiens

<400> 654
Met Pro Leu Phe Leu Phe Val Ala His Leu Ile Ser Leu Leu Leu Ala
1 5 10 15

Phe Arg Arg Pro Pro Ala Ser Gln Ile Thr Pro Arg Ala Trp Thr Thr 20 25 30

Glu Ile Ala Ser Cys Glu Ser Val Glu Met Val Lys Ala Leu Ser Ser 35 40 45

Leu Arg Ser Arg Ala Gln Val Asn Ala Asp Phe Pro Gly His Leu Cys 50 60

<210> 655

<211> 385

<212> PRT

<213> Homo sapiens

<400> 655

Met Ser Phe Ile Met Lys Leu His Arg His Phe Gln Arg Thr Val Ile 1 5 10 15

Leu Leu Ala Thr Phe Cys Met Val Ser Ile Ile Ser Ala Tyr Tyr 20 25 30

Leu Tyr Ser Gly Tyr Lys Gln Glu Asn Glu Leu Ser Glu Thr Ala Ser 35 40 45

Glu Val Asp Cys Gly Asp Leu Gln His Leu Pro Tyr Gln Leu Met Glu 50 55 60

Val Lys Ala Met Lys Leu Phe Asp Ala Ser Arg Thr Asp Pro Thr Val 65 70 75 80

Leu Val Phe Val Glu Ser Gln Tyr Ser Ser Leu Gly Gln Asp Ile Ile 85 90 95

Met Ile Leu Glu Ser Ser Arg Phe Gln Tyr His Ile Glu Ile Ala Pro 100 105 110

Gly Lys Gly Asp Leu Pro Val Leu Ile Asp Lys Met Lys Gly Lys Tyr 115 120 125

Ile Leu Ile Ile Tyr Glu Asn Ile Leu Lys Tyr Ile Asn Met Asp Ser 130 135 140

Trp Asn Arg Ser Leu Leu Asp Lys Tyr Cys Val Glu Tyr Gly Val Gly 145 150 155 160

Val Ile Gly Phe His Lys Thr Ser Glu Lys Ser Val Gln Ser Phe Gln 165 170 175

Leu Lys Gly Phe Pro Phe Ser Ile Tyr Gly Asn Leu Ala Val Lys Asp 180 185 190

Cys Cys Ile Asn Pro His Ser Pro Leu Ile Arg Val Thr Lys Ser Ser 195 200 205

Lys Leu Glu Lys Gly Ser Leu Pro Gly Thr Asp Trp Thr Val Phe Gln 210 215

Ile Asn His Ser Ala Tyr Gln Pro Val Ile Phe Ala Lys Val Lys Thr

225 230 235 240

Pro Glu Asn Leu Ser Pro Ser Ile Ser Lys Gly Ala Phe Tyr Ala Thr 245 250 255

Ile Ile His Asp Leu Gly Leu His Asp Gly Ile Gln Arg Val Leu Phe 260 265 270

Gly Asn Asn Leu Asn Phe Trp Leu His Lys Leu Ile Phe Ile Asp Ala 275 280 285

Ile Ser Phe Leu Ser Gly Lys Arg Leu Thr Leu Ser Leu Asp Arg Tyr 290 295 300

Ile Leu Val Asp Ile Asp Asp Ile Phe Val Gly Lys Glu Gly Thr Arg 305 310 315 320

Met Asn Thr Asn Asp Val Lys Val Arg Leu Tyr Phe Leu Lys Phe Gln 325 330 335

Ser Ser Val His Leu Pro Ala Gly Ile Gln Leu Ser Gln Phe Val Leu 340 345 350

Gln Leu Gly Tyr Pro Gly His Gly Ile Tyr Trp Glu Ser Leu Gly Asn \$355\$

Leu Gly Leu Ser Leu Thr Leu Asn Gln Leu Arg Arg Leu Cys Ile Ser 370 380

Ile 385

<210> 656

<211> 53

<212> PRT

<213> Homo sapiens

<400> 656

Met Leu Val Phe Leu Leu Phe Ser Thr Val Thr Val Leu Cys Leu
1 5 10 15

Lys Val Val Phe Ser Leu Lys Ala Val Ala Tyr Ile Val Lys Asn Glu 20 25 30

Gly Leu Cys Leu Lys Phe Ile Ala Leu Gln Arg Val Val Ser Leu Lys $35 \hspace{1cm} 40 \hspace{1cm} 45$

Ser Cys Thr Ile Lys 50

<210> 657

<211> 49

<212> PRT

<213> Homo sapiens

<400> 657

Met Asn Leu Leu Gly Met Ile Phe Ser Met Cys Gly Leu Met Leu Lys

1 5 10 15

Leu Lys Trp Cys Ala Trp Val Ala Val Tyr Cys Ser Phe Ile Ser Phe 20 25 30

Ala Asn Ser Arg Ser Ser Glu Asp Thr Lys Gln Met Met Ser Ser Phe 35 40 45

Met

<210> 658 <211> 110 <212> PRT <213> Homo sapiens

Asp Val Glu Ala Leu Ala Ser Leu Pro Glu Asp Arg Leu Arg Trp Asn 20 25 30

Leu Leu Ala Leu Pro Ala Ser Pro Cys Ala Val Thr Ala Leu Val Ala 35 40 45

Arg His Arg Arg Ala Gly Leu Gln Arg Ser Ile Gln Cys Leu Leu Gly 50 60

Arg Gln Gly Gly Gly Cys Asn Cys Glu Leu Thr Lys Pro Gln Val 65 70 75 80

Gly Ser Lys Trp Val Gly His Arg Lys Lys Ser Asp Leu Gln Ser Gly 85 90 95

Asp Leu Gly Ser Gly Leu Cys Leu Met Thr Gly Ser Val Met 100 $$105\$

<210> 659 <211> 258 <212> PRT <213> Homo sapiens

Met Tyr Ile Trp Phe Ile Ile Phe Phe Ile Gln Pro His Lys Glu Glu

1 5 10 15

Arg Phe Leu Phe Pro Val Tyr Pro Leu Ile Cys Leu Cys Gly Ala Val 20 25 30

Ala Leu Ser Ala Leu Gln Lys Cys Tyr His Phe Val Phe Gln Arg Tyr 35 40 45

Arg Leu Glu His Tyr Thr Val Thr Ser Asn Trp Leu Ala Leu Gly Thr 50 60

Val Phe Leu Phe Gly Leu Leu Ser Phe Ser Arg Ser Val Ala Leu Phe

65 70 75 80

Arg Gly Tyr His Gly Pro Leu Asp Leu Tyr Pro Glu Phe Tyr Arg Ile $85 \hspace{1.5cm} 90 \hspace{1.5cm} 95$

Ala Thr Asp Pro Thr Ile His Thr Val Pro Glu Gly Arg Pro Val Asn 100 \$105

Val Cys Val Gly Lys Glu Trp Tyr Arg Phe Pro Ser Ser Phe Leu Leu 115 120 125

Pro Asp Asn Trp Gln Leu Gln Phe Ile Pro Ser Glu Phe Arg Gly Gln 130 135 140

Leu Pro Lys Pro Phe Ala Glu Gly Pro Leu Ala Thr Arg Ile Val Pro 145 150 155 160

Thr Asp Met Asn Asp Gln Asn Leu Glu Glu Pro Ser Arg Tyr Ile Asp 165 170 175

Ile Ser Lys Cys His Tyr Leu Val Asp Leu Asp Thr Met Arg Glu Thr 180 185 190.

Pro Arg Glu Pro Lys Tyr Ser Ser Asn Lys Glu Glu Trp Ile Ser Leu 195 200 205

Ala Tyr Arg Pro Phe Leu Asp Ala Ser Arg Ser Ser Lys Leu Leu Arg 210 215 220

Ala Phe Tyr Val Pro Phe Leu Ser Asp Gln Tyr Thr Val Tyr Val Asn 225 230 235 240

Tyr Thr Ile Leu Lys Pro Arg Lys Ala Lys Gln Ile Arg Lys Lys Ser 245 250 255

Gly Gly

<210> 660

<211> 59

<212> PRT

<213> Homo sapiens

<400> 660

Met Asn Ser Thr Leu Cys Val Val Leu Ser Leu Met Cys Met Asn Ser

Thr Leu Cys Val Val Leu Ser Leu Thr His Ser Cys Pro Ser Pro Gln 20 25 30

Val Pro Lys Val His Tyr Met Ile Phe Met Pro Leu His Leu His Ser 35 40 45

Leu Ala Leu Thr Gln Leu Ile Ile Tyr Lys
50

<210> 661

<211> 202

<212> PRT

<213> Homo sapiens

<400> 661

Met Ser Leu Leu Val Asp Gly Asp Met Asn Leu Ser Ile Ile Met Thr 1 5 10 15

Ile Ser Ser Thr Leu Leu Ala Leu Val Leu Met Pro Leu Cys Leu Trp 20 25 30

Ile Tyr Ser Trp Ala Trp Ile Asn Thr Pro Ile Val Gln Leu Leu Pro $35 \hspace{1cm} 40 \hspace{1cm} 45$

Leu Gly Thr Val Thr Leu Thr Leu Cys Ser Thr Leu Ile Pro Ile Gly 50 60

Leu Gly Val Phe Ile Arg Tyr Lys Tyr Ser Arg Val Ala Asp Tyr Ile 65 70 75 80

Val Lys Val Ser Leu Trp Ser Leu Leu Val Thr Leu Val Val Leu Phe
85 90 95

Ile Met Thr Gly Thr Met Leu Gly Pro Glu Leu Leu Ala Ser Ile Pro
100 105 110

Ala Ala Val Tyr Val Ile Ala Ile Phe Met Pro Leu Ala Gly Tyr Ala 115 120 125

Ser Gly Tyr Gly Leu Ala Thr Leu Phe His Leu Pro Pro Asn Cys Lys 130 135 140

Arg Thr Val Cys Leu Glu Thr Gly Ser Gln Asn Val Gln Leu Cys Thr 145 150 155 160

Ala Ile Leu Lys Leu Ala Phe Pro Pro Gln Phe Ile Gly Ser Met Tyr 165 170 175

Met Phe Pro Leu Leu Tyr Ala Leu Phe Gln Ser Ala Glu Ala Gly Ile 180 185 190

Phe Val Leu Ile Tyr Lys Met Tyr Gly Arg 195 200

<210> 662

<211> 80

<212> PRT

<213> Homo sapiens

<400> 662

Met Ala Leu Gly Ser Met Tyr Leu Val Leu Thr Leu Ile Val Ala Lys
1 5 10 15

Val Leu Arg Gly Ala Glu Pro Cys Cys Gly Pro Leu Lys Asn Arg Val 20 25 30

Leu Arg Pro Cys Pro Leu Pro Val His Cys Pro Leu Pro Ile Pro Ser 35 40 45

Pro Ala Glu Gly Ile Pro Trp Val Ala Tyr Leu Pro Ile Arg Trp Phe 50 60

Ile Ser Cys Cys Pro Gly His Cys Ile Gln Ile Pro Met Cys Thr Ser 65 70 75 80

<210> 663

<211> 240

<212> PRT

<213> Homo sapiens

<400> 663

Met Gly Asn Cys Gln Ala Gly His Asn Leu His Leu Cys Leu Ala His 1 5 10 15

His Pro Pro Leu Val Cys Ala Thr Leu Ile Leu Leu Leu Gly Leu 20 25 30

Ser Gly Leu Gly Leu Gly Ser Phe Leu Leu Thr His Arg Thr Gly Leu 35 40 45

Arg Ser Pro Asp Ile Pro Gln Asp Trp Val Ser Phe Leu Arg Ser Phe 50 55 60

Gly Gln Leu Thr Leu Cys Pro Arg Asn Gly Thr Val Thr Gly Lys Trp 65 70 75 80

Arg Gly Ser His Val Val Gly Leu Leu Thr Thr Leu Asn Phe Gly Asp 85 90 95

Gly Pro Asp Arg Asn Lys Thr Arg Thr Phe Gln Ala Thr Val Leu Gly 100 105 110

Ser Gln Met Gly Leu Lys Gly Ser Ser Ala Gly Gln Leu Val Leu Ile 115 120 125

Thr Ala Arg Val Thr Thr Glu Arg Thr Ala Gly Thr Cys Leu Tyr Phe 130 135 140

Ser Ala Val Pro Gly Ile Leu Pro Ser Ser Gln Pro Pro Ile Ser Cys 145 150 155 160

Ser Glu Glu Gly Ala Gly Asn Ala Thr Leu Ser Pro Arg Met Gly Glu 165 170 175

Glu Cys Val Ser Val Trp Ser His Glu Gly Leu Val Leu Thr Lys Leu 180 185 190

Leu Thr Ser Glu Glu Leu Ala Leu Cys Gly Ser Arg Leu Leu Val Leu 195 200 205

Gly Ser Phe Leu Leu Phe Cys Gly Leu Leu Cys Cys Val Thr Ala 210 215 220

Met Cys Phe His Pro Arg Arg Glu Ser His Trp Ser Arg Thr Arg Leu 225 230 235 240

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<210> 664
<211> 39
<212> PRT
<213> Homo sapiens
<400> 664
Met Leu Leu Leu Lys Thr Leu Phe Val Thr Phe Trp Ser Thr Asn
                                    10
Leu Ser Ile Thr Phe Ser Asn Tyr Asn Val Lys Leu Tyr Gln Trp Gln
Ser Tyr Ile Val Asn Gly Ser
<210> 665
<211> 146
<212> PRT
<213> Homo sapiens
<400> 665
Met Leu Met Pro Val His Phe Leu Leu Leu Leu Leu Leu Leu Gly
                                   10
Gly Pro Arg Thr Gly Leu Pro His Lys Phe Tyr Lys Ala Lys Pro Ile
Phe Ser Cys Leu Asn Thr Ala Leu Ser Glu Ala Glu Lys Gly Gln Trp
                            40
Glu Asp Ala Ser Leu Leu Ser Lys Arg Ser Phe His Tyr Leu Arg Ser
                       55
Arg Asp Ala Ser Ser Gly Glu Glu Glu Glu Gly Lys Glu Lys Lys Thr
Phe Pro Ile Ser Gly Ala Arg Gly Gly Ala Arg Gly Thr Arg Tyr Arg
Tyr Val Ser Gln Ala Gln Pro Arg Gly Lys Pro Arg Gln Asp Thr Ala
Lys Ser Pro His Arg Thr Lys Phe Thr Leu Ser Leu Asp Val Pro Thr
Asn Ile Met Asn Leu Leu Phe Asn Ile Ala Lys Ala Lys Asn Leu Arg
Ala Gln
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145

<210> 666

<211> 174

<212> PRT

<213> Homo sapiens

<400> 666

Met Glu Ala Pro Gly Pro Arg Ala Leu Arg Thr Ala Leu Cys Gly Gly 1 5 10 15

Cys Cys Cys Leu Leu Cys Ala Gl
n Leu Ala Val Ala Gly Lys Gly 20 25 30

Ala Arg Gly Phe Gly Arg Gly Ala Leu Ile Arg Leu Asn Ile Trp Pro 35 40

Ala Val Gln Gly Ala Cys Lys Gln Leu Glu Val Cys Glu His Cys Val
50 55 60

Glu Gly Asp Arg Ala Arg Asn Leu Ser Ser Cys Met Trp Glu Gln Cys 65 70 75 80

Arg Pro Glu Glu Pro Gly His Cys Val Ala Gln Ser Glu Val Val Lys
85 90 95

Glu Gly Cys Ser Ile Tyr Asn Arg Ser Glu Ala Cys Pro Ala Ala His 100 105 110

His His Pro Thr Tyr Glu Pro Lys Thr Val Thr Thr Gly Ser Pro Pro 115 120 125

Val Pro Glu Ala His Ser Pro Gly Phe Asp Gly Ala Ser Phe Ile Gly
130 135 140

Gly Val Val Leu Val Leu Ser Leu Gln Ala Val Ala Phe Phe Val Leu 145 150 155 160

His Phe Leu Lys Ala Lys Asp Ser Thr Tyr Gln Thr Leu Ile 165 170

<210> 667

<211> 199

<212> PRT

<213> Homo sapiens

<400> 667

Met Arg Arg Leu Leu Leu Ala Leu Pro Phe Ala Leu Leu Pro Leu Ala 1 5 10 15

Val Ala His Ala His Glu Asp His Asp His Glu His Gly Ser Leu Gly 20 25 30

Ala His Glu His Gly Val Gly Arg Leu Asn Ala Val Leu Asp Gly Gln 35 40 45

Ala Leu Glu Leu Glu Leu Asp Ser Pro Ala Met Asn Leu Val Gly Phe 50 60

Glu His Val Ala Thr Ser Ala Ala Asp Lys Ala Lys Val Ala Ala Val 65 70 75 80

Arg Lys Gln Leu Glu Asn Pro Ser Ala Leu Phe Asn Leu Pro Lys Ala 85 90 95

Ala Gly Cys Val Val Ser Ser Gln Glu Leu Asn Ser Pro Leu Phe Gly 100 105 110

Asp Lys Pro Glu Ala Glu His Asp Asp Asp His Ala Ser Asp Gly 115 120 125

Lys Gly Ala Ala Ala His Lys His Asp His Asp His Ser Glu Ile His 130 135 140

Ala His Tyr Gln Phe Thr Cys Ala Thr Pro Thr Ala Leu Gly Asn Leu 145 150 155 160

Asp Leu Ser Gln Val Phe Lys Thr Phe Pro Ala Thr Gln Lys Ile Gln 165 170 175

Val Gln Leu Ile Gly Pro Ser Gly Gln Gln Gly Val Asp Ala Thr Ala 180 185 190

Thr Ala Ala Thr Leu Lys Phe 195

<210> 668

<211> 84

<212> PRT

<213> Homo sapiens

<220>

<221> SITE

<222> (65)

<223> Xaa equals any amino acid

<400> 668

Met Trp Val Phe Phe Leu Pro Phe Phe Ser Ile Leu Phe Lys Ile Cys 1 10 15

Trp Cys Ile Ser Leu Ser Gln Thr Lys Glu Lys Gln Ser Ser Asn Leu 20 25 30

Met Phe Tyr Phe Phe Cys Ile Cys Thr Tyr Glu Arg Arg Lys Lys 35 40 45

Glu Met Arg Arg Gly Glu Lys Lys Arg Ser Phe Cys Leu Ile Gly Leu 50 60

Xaa Gln His Met Ile Ala Val Gln Ala Trp Phe His Glu Gln His Gln 65 70 75 80

Ile Gln Ile Ser

<210> 669

<211> 245

<212> PRT

<213> Homo sapiens

<400> 669

Met Glu Gly Pro Arg Gly Trp Leu Val Leu Cys Val Leu Ala Ile Ser 1 5 10 15

Leu Ala Ser Met Val Thr Glu Asp Leu Cys Arg Ala Pro Asp Gly Lys 20 25 30

Lys Gly Glu Ala Gly Arg Pro Gly Arg Arg Gly Arg Pro Gly Leu Lys $35 \hspace{1cm} 40 \hspace{1cm} 45$

Gly Glu Gln Gly Glu Pro Gly Ala Pro Gly Ile Arg Thr Gly Ile Gln 50 60

Gly Leu Lys Gly Asp Gln Gly Glu Pro Gly Pro Ser Gly Asn Pro Gly 65 70 75 80

Lys Val Gly Tyr Pro Gly Pro Ser Gly Pro Leu Gly Ala Arg Gly Ile 85 90 95

Pro Gly Ile Lys Gly Thr Lys Gly Ser Pro Gly Asn Ile Lys Asp Gln 100 \$105

Pro Arg Pro Ala Phe Ser Ala Ile Arg Arg Asn Pro Pro Met Gly Gly 115 120 125

Asn Val Val Ile Phe Asp Thr Val Ile Thr Asn Gln Glu Glu Pro Tyr 130 135 140

Gln Asn His Ser Gly Arg Phe Val Cys Thr Val Pro Gly Tyr Tyr Tyr 145 150 155 160

Phe Thr Phe Gln Val Leu Ser Gln Trp Glu Ile Cys Leu Ser Ile Val 165 170 175

Ser Ser Ser Arg Gly Gln Val Arg Arg Ser Leu Gly Phe Cys Asp Thr 180 185 190

Thr Asn Lys Gly Leu Phe Gln Val Val Ser Gly Gly Met Val Leu Gln 195 200 205

Leu Gln Gln Gly Asp Gln Val Trp Val Glu Lys Asp Pro Lys Lys Gly 210 215 220

His Ile Tyr Gln Gly Ser Glu Ala Asp Ser Val Phe Ser Gly Phe Leu 225 230 235 240

Ile Phe Pro Ser Ala 245

<210> 670

<211> 83

<212> PRT

<213> Homo sapiens

<400> 670

Met Gly Gln Cys Pro Gly Ser Arg Val Leu Pro Gln Leu Met Gln Leu 1 5 10 15

Trp Leu Leu Cys Ala Gln Ile Met Cys Leu Glu Ala Phe Leu Gln 20 25 30

Gln Gly Ser Val Arg Lys Trp Lys Ser Gly Val Ser Ser Phe Pro Gly 35 40 45

Glu Ser Leu Ala Glu Gln Leu Thr Leu Ser Lys His Cys Arg Trp Pro 50 60

Leu Phe Leu Pro Gly Ser Ser Ser Trp Glu Leu Ser Ala Pro Gly Lys
65 70 75 80

Phe Trp Gln

<210> 671

<211> 61

<212> PRT

<213> Homo sapiens

<400> 671

Met Tyr Leu Phe Leu Lys Thr Leu Leu Ser Phe Ser Thr Leu Met Met 1 5 10 15

Thr Thr Ala Leu Ser Phe Met Val Ile Thr Val Leu Trp Val Leu Leu 20 25 30

Leu His Leu Leu Ala Asn Ile Cys Ile Pro Arg Lys Cys Ser Phe Ala $35 \hspace{1.5cm} 40 \hspace{1.5cm} 45$

Cys Phe Tyr Ile Asn Gly Ile Leu Leu His Ala Val Phe 50 60

<210> 672

<211> 57

<212> PRT

<213> Homo sapiens

<400> 672

Met Ala Val Ser Val Ile Phe Cys Gln Lys Leu Lys Thr Gly Ser Val 1 5 10 15

Lys Leu Trp Ile Gln Met Leu Leu Trp Leu Gln Phe Ser Val Ala Cys $20 \hspace{1cm} 25 \hspace{1cm} 30$

Leu Arg Leu Arg Lys Gly Gly Lys Trp Ser Pro Trp Gly Leu Met Leu 35 40 45

Lys Glu Val Ile Trp Lys Asp Cys Arg 50 55

<210> 673

<211> 83

<212> PRT

<213> Homo sapiens

<400> 673

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Trp Val Ala Leu Gly Glu Asp Tyr Leu Gly Thr Pro Ile Leu Ile Pro
35 40 45

Asn Ile His Gln Thr Cys Pro His Pro Pro Leu Trp Glu Leu Val Pro 50 55 60

Glu His Pro Cys Arg Leu Val Leu Ile Phe Ser Leu Cys Glu His Thr 65 70 75 80

His Ile Arg

<210> 674

<211> 56

<212> PRT

<213> Homo sapiens

<400> 674

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Cys Thr Glu Leu Trp Lys Gly Ala Pro Cys Arg His Ile Phe Gln

Thr Gly Pro Asp Leu Leu Val Thr Gln Arg Cys Val His Ser Leu Leu 35 40 45

Leu Gly Tyr Leu Ile Ser Ile Phe 50 55

<210> 675

<211> 319

<212> PRT

<213> Homo sapiens

<400> 675

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Leu Leu Arg Leu Arg Pro Val Phe Ser Pro Leu His Ser Gly Pro Gly 35 40

Lys Pro Ala Gln Phe Leu Ala Gly Glu Ala Glu Glu Val Asn Ala Phe 50 55 60

Ala Leu Gly Phe Leu Ser Thr Ser Ser Gly Val Ser Gly Glu Asp Glu 70 Val Glu Pro Leu His Asp Gly Val Glu Glu Ala Glu Lys Lys Met Glu Glu Glu Gly Val Ser Val Ser Glu Met Glu Ala Thr Gly Ala Gln Gly Pro Ser Arg Val Glu Glu Ala Glu Gly His Thr Glu Val Thr Glu Ala Glu Gly Ser Gln Gly Thr Ala Glu Ala Asp Gly Pro Gly Ala Ser Ser Gly Asp Glu Asp Ala Ser Gly Arg Ala Ala Ser Pro Glu Ser Ala Ser Ser Thr Pro Glu Ser Leu Gln Ala Arg Arg His His Gln Phe Leu Glu 170 Pro Ala Pro Ala Pro Gly Ala Ala Val Leu Ser Ser Glu Pro Ala Glu Pro Leu Leu Val Arg His Pro Pro Arg Pro Arg Thr Thr Gly Pro Arg Pro Arg Gln Asp Pro His Lys Ala Gly Leu Ser His Tyr Val Lys Leu Phe Ser Phe Tyr Ala Lys Met Pro Met Glu Arg Lys Ala Leu Glu Met Val Glu Lys Cys Leu Asp Lys Tyr Phe Gln His Leu Cys Asp Asp Leu Glu Val Phe Ala Ala His Ala Gly Arg Lys Thr Val Lys Pro Glu Asp Leu Glu Leu Leu Met Arg Arg Gln Gly Leu Val Thr Asp Gln Val Ser 275 280 285 Leu His Val Leu Val Glu Arg His Leu Pro Leu Glu Tyr Arg Gln Leu Leu Ile Pro Cys Ala Tyr Ser Gly Asn Ser Val Phe Pro Ala Gln 310

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<211> 336

<212> PRT

<213> Homo sapiens

<400> 676

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Val Tyr His Glu Leu Ile Gln Arg Met Tyr Thr Arg Leu Glu Pro Leu 20 25 30

			such as T-cells).	response element activity of polypeptides of the invention (including antibodies and agonists or
				and gonists of the invention) include assays disclosed in Berger et al., Gene 60:1-10 (1998); Cullen and Malm Methods in Enzymol 216:362-368 (1992). Henthom et al., Proc Natl Acad Sci 11SA
				85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-
				844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T
				cells that may be used according to these assays are publicly available (e.g., through the ATCC).
 -				Exemplary human T cells that may be used according to these assays include the SUPT cell line,
				which is a suspension culture of IL-2 and IL-4 responsive T cells.
75	HDPCY37	200	Production of IL-	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used
			10 and activation	or routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			of T-cells.	agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation
				of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of
				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
				modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed
				and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-
_				968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology &
				Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
				reference in their entirety. Exemplary cells that may be used according to these assays include Th2
				cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells
				are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation
				and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and
				asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions
				using peripheral blood lymphocytes isolated from cord blood.
9/	HDPFB02	701	Activation of	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast
_			transcription	cell line. Activation of GATA:3 in mast cells has been linked to cytokine and chemokine
			through GATA-3	production. Assays for the activation of transcription through the GATA3 response element are
			response element	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
			in immune cells	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
			(such as mast	transcription factors and modulate expression of mast cell genes important for immune response
			cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test GATA3-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold

				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available
_				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
9/	HDPFB02	701	Production of	Endothelial cells, which are cells that line blood vessels, and are involved in functions that include,
			ICAM in	but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell
			endothelial cells	extravasation. Exemplary endothelial cells that may be used in ICAM production assays include
			(such as human	human umbilical vein endothelial cells (HUVEC), and are available from commercial sources. The
			umbilical vein	expression of ICAM (CD54), a intergral membrane protein, can be upregulated by cytokines or other fortures and ICAM expression is important in mediating immine and endothelial call interactions
			-	tactors, and texture on find amount to make the formation of the first of the first of the Market of
			(())	reading to infinition and initialization of responses. Assays for incasuring expression of incarnations of well-known in the set and may be used or routinely modified to access the ability of notwentides of
				the invention (including antibodies and appnists of antagonists of the invention) to regulate ICAM-1
				expression. Exemplary assays that may be used or routinely modified to measure ICAM-1
				expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000);
				Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J
				Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein
				incorporated by reference in its entirety.
11	HDPFF39	702	Activation of T-	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation,
			Cell p38 or JNK	activation, or apoptosis are well known in the art and may be used or routinely modified to assess
			Signaling	the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			Pathway.	invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis.
				Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test
				JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and
				agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem
				379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM,
-				Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb
				MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein
				incorporated by reference in its entirety. T cells that may be used according to these assays are
				publicly available (e.g., through the ATCC). Exemplary mouse 1 cells that may be used according

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				to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with evtotoxic activity.
. 78	HDPFP29	703	Myoblast cell proliferation	Assays for muscle cell proliferation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit myoblast cell proliferation. Exemplary assays for myoblast cell proliferation that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays disclosed in: Soeta, C., et al. "Possible role for the c-ski gene in the proliferation of myogenic cells in regenerating skeletal muscles of rats" Dev Growth Differ Apr;43(2):155-64 (2001); Ewton DZ, et al., "IGF binding proteins-4, -5 and -6 may play specialized roles during L6 myoblast proliferation and differentiation." J Endocrinol Mar; 144(3):539-53 (1995); and, Pampusch MS, et al., "Effect of transforming growth factor beta on proliferation of L6 and embryonic porcine myogenic cells." J Cell Physiol Jun; 143(3):524-8 (1990); the contents of each of which are herein incorporated by reference in their entirety. Exemplary myoblast cells that may be used according to these assays include the rat myoblast L6 cell line. Rat myoblast L6 cells are an adherent rat myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuse to form multinucleated myotubes and striated fibers after culture in differentiation media.
79	HDPGT01	704	Regulation of transcription through the FAS promoter element in hepatocytes	Assays for the regulation of transcription through the FAS promoter element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis. FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays, such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.
80	HDPHI51	705	Regulation of	Assays for the regulation of transcription through the FAS promoter element are well-known in the

		1
art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis. FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Methods in Enzymol. 216:362–368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays, such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in
transcription through the FAS promoter element in hepatocytes	Activation of transcription through STAT6 response element in immune cells (such as T-cells).	Activation of transcription through NFAT
	705	902
	HDPHIS1	HDPJF37
	08	81

	(such	immune cells (such as T-cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available
Production of	ji	jo uoi	(e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated. MCP-1 FMAT. Assays for immunomodulatory proteins that are produced by a large variety of cells and set to induce the material produced by a large variety of cells and set to induce the material produced by a large variety of cells are produced by a large variety of cells and set to induce the material produced by a large variety of cells are to induce the material produced by a large variety of the material produced by a large variety of the material produced by a large variety of the material produced by a large variety of the material produced by a large variety of the material produced by a large variety of the m
	-		may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce chemotaxis, and modulate immune cell activation. Exemplary assays that test for immunomodulatory proteins evaluate the production of cell surface markers, such as monocyte chemoattractant protein (MCP), and the activation of monocytes and T cells. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
			include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.
Regulation of transcription through the FAS	llatic cript gh t	on of tion he FAS	Assays for the regulation of transcription through the FAS promoter element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter
promoter element in hepatocytes	oter e	lement ytes	element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis. FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS

				gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat
				glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS
				promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies
				and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl
				Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999);
				Oskouian B, et al., Biochem J. 317 (Pt 1):257-65 (1996); Berger, et al., Gene 66:1-10 (1988); and,
				Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein
				incorporated by reference in its entirety. Hepatocytes that may be used according to these assays,
				such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely
				generated. Exemplary hepatocytes that may be used according to these assays include rat liver
				hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.
83	HDPMM88	708	Myoblast cell	Assays for muscle cell proliferation are well known in the art and may be used or routinely modified
			proliferation	to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to stimulate or inhibit myoblast cell proliferation. Exemplary assays
				for myoblast cell proliferation that may be used or routinely modified to test activity of polypeptides
				and antibodies of the invention (including agonists or antagonists of the invention) include, for
				example, assays disclosed in: Soeta, C., et al. "Possible role for the c-ski gene in the proliferation of
				myogenic cells in regenerating skeletal muscles of rats" Dev Growth Differ Apr;43(2):155-64
				(2001); Ewton DZ, et al., "IGF binding proteins-4, -5 and -6 may play specialized roles during L6
				myoblast proliferation and differentiation" J Endocrinol Mar;144(3):539-53 (1995); and, Pampusch
				MS, et al., 'Effect of transforming growth factor beta on proliferation of L6 and embryonic porcine
				myogenic cells" J Cell Physiol Jun;143(3):524-8 (1990); the contents of each of which are herein
				incorporated by reference in their entirety. Exemplary myoblast cells that may be used according to
				these assays include the rat myoblast L6 cell line. Rat myoblast L6 cells are an adherent rat
				myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuse to form
				multinucleated myotubes and striated fibers after culture in differentiation media.
84	HDPOE32	709	Production of	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely
			ICAM-1	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or
				routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al,
				FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000),
				the contents of each of which is herein incorporated by reference in its entirety. Cells that may be
				used according to these assays are publicly available (e.g., through the ATCC) and/or may be
				routinely generated. Exemplary cells that may be used according to these assays include

				microvascular endothelial cells (MVEC).
85	нрроно6	710	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or
				routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al,
				FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000),
				the contents of each of which is never incorporated by reference in its entirety. Cells that may be
				used according to these assays are publicly available (e.g., through the ATCC) and/or may be
				rounlety generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
85	НДРОН06	710	Production of IL-	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used
			10 and activation	or routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			of T-cells.	agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation
				of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of
				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
				modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed
		-		and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-
				968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology $\&$
				Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
				reference in their entirety. Exemplary cells that may be used according to these assays include Th2
				cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells
				are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation
				and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and
				asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions
				using peripheral blood lymphocytes isolated from cord blood.
98	HDPOJ08	711	Regulation of	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or
			apoptosis in	routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			pancreatic beta	agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis.
			cells.	Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary
				assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity
				of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et
				al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):687-
				94 (2000); Chandra J, et al., Diabetes, 30 Suppl 1:344-7 (2001); Suk R, et al., J Innnunol,

106(7):4481-9 (2001); Tejedo J, et al., FEBS Lett, 459(2):238-43 (1999); Zhang, S., et al., FEBS Lett, 458(2-3): 122-125 (2000); Not et al., 1 Vasc Res 37(3): 209-218 (2000); and Kassan and Harlan. J Atheroscler Thromb 3(2): 7-58 (1996); the contents of each of which are herein incoporated by reference in its entirety. Pancreatic cells that may be used according to these assays a reclude MR.m. R.H.m. is a tat adheren pancraatic bate of line derived from a radiation included transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide hormones, and produce insulin. somatostatin, and possibly glucagon. ATTC. #CRL.2057 Chick et al. Proc. Natl. Acad. Sci. 1997 74:628. AF et al. Proc. Natl. Acad. Sci. 1997 74:628. AF et al. Proc. Natl. Acad. Sci. 1997 74:628. AF et al. Proc. Natl. Acad. Sci. 1997 74:628. AF et al. Proc. Natl. Acad. Sci. 1997 74:628. AF et al. Proc. Natl. Acad. Sci. 1997 74:628. AF et al. Proc. Natl. Acad. Sci. 1997 74:628. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:629. AF et al. Proc. Natl. Acad. Sci. 1997 74:409. AF et al. Proc. Natl. Acad. Sci. 1997 74:409. AF et al. Proc. Natl. Acad. Sci. 1997 74:409. AF et al. Proc. Natl. Acad. Sci. 1997 74:409. AF et al. Respired to the scassy that may be used or routinely modified to use for simulation of agonists or antagonists of the invention in incorporated by reference in its entirety. Parcreatic cells that may be used according to these assays i
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HDPPN86

			agonists of antagonists of the invention) include assays disclosed in: Amen, b., et al., Am J Fnyslol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al.,
			FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-
		-	204 (1999), the contents of each of which is herein incorporated by reference in its entirety.
		•	Pancreatic cells that may be used according to these assays are publicly available (e.g., through the
			ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according
			to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from
			cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics
		•	typical of native pancreatic beta cells including glucose inducible insulin secretion. References:
			Asfari et al. Endocrinology 1992 130:167.
HDPSB18	713	Production of IL-	IL-10 FMAT. Assays for immunomodulatory proteins produced by activated T cells, B cells, and
		10 and	monocytes that exhibit anti-inflammatory activity and downregulate monocyte/macrophage function
		downregulation of	and expression of cytokines are well known in the art and may be used or routinely modified to
<u> </u>		immune responses	assess the ability of the polypeptides of the invention (including antibodies and agonists or
			antagonists of the invention) to mediate immunomodulation, regulate inflammatory activities, and
			modulate immune cell function and cytokine production. Exemplary assays that test for
			immunomodulatory proteins evaluate the production of cytokines, such as IL-10, and the
			downmodulation of immune responses. Such assays that may be used or routinely modified to test
			immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or
			antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular
			Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-
			160 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are
			herein incorporated by reference in its entirety. Human T cells that may be used according to these
			assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T
			cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and
			CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be
			preactivated to enhance responsiveness to immunomodulatory factors.
HDPSH53	714	Stimulation of	Assays for measuring calcium flux are well-known in the art and may be used or routinely
		Calcium Flux in	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
		pancreatic beta	antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to
		cells.	measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium
			compared to much higher extracellular calcium. Extracellular factors can cause an influx of
			calcium, leading to activation of calcium responsive signaling pathways and alterations in cell
_			functions. Exemplary assays that may be used or routinely modified to measure calcium flux by

	-			polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995); Mogami H, et al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
06	HDPSP01	715	Production of MCP-1	MCP-1 FMAT. Assays for immunomodulatory proteins that are produced by a large variety of cells and act to induce chemotaxis and activation of monocytes and T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation (including antibodies and agonists or antagonists of the invention. Exemplary assays that test for immunomodulatory proteins evaluate the production of cell surface markers, such as monocyte chemoattractant protein (MCP), and the activation of monocytes and T cells. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.
06	HDPSP01	715	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component

in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of	each of which is herein incorporated by reference in its entirety. Fancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity
		Activation of Endothelial Cell JNK Signaling Pathway.	Regulation of apoptosis in pancreatic beta cells.
		716	716
		HDPSP54	HDPSP54
		91	91

of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):687-94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:S44-7 (2001); Suk K, et al., J Immunol, 166(7):4481-9 (2001); Tejedo J, et al., FEBS Lett, 455(2):238-43 (1999); Zhang, S., et al., FEBS Lett, 455(3):315-20 (1999); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a radiation induced transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1980 77:3519.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics, 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL.10, IL.13, IL.5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK
	Production of IL- 10 and activation of T-cells.	Activation of Adipocyte ERK Signaling Pathway
	716	717
	HDPSP54	HDPUW68
·	91	92

kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.	Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995);Mogami H, et al., Endocrinology, 136(7):2960-6 (1995): Richardson SB, et al., Biochem J. 288 (Pt 3):847-51
	Activation of transcription through serum response element in immune cells (such as T-cells).	Stimulation of Calcium Flux in pancreatic beta cells.
	717	717
	HDPUW68	HDPUW68
	92	93

each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Kinase assay. Kinase assays, for example an GSK-3 kinase assay, for PI3 kinase signal transduction that regulate glucose metabolism and cell survivial are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit glucose metabolism and cell survival. Exemplary assays for PI3 kinase activity that may be used or routinely modified to test PI3 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Nikoulina et al., Diabetes 49(2):263-271 (2000); and Schreyer et al., Diabetes 48(8):1662-1666 (1999), the contents of each of which are herein incorporated by reference in its entirety. Rat myoblast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary rat myoblast cells that may be used according to these assays include L6 cells. L6 is an adherent rat myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuses to form multinucleated myotubes and striated fibers after culture in differentiation media.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
	Activation of Skeletal Mucle Cell PI3 Kinase Signalling Pathway	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).
	717	718
	HDPUW68	HDPVW11
	92	93

late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" Clin Exp Immunol; Oct; 122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and
	Activation of Endothelial Cell p38 or JNK Signaling Pathway.	Insulin Secretion
	719	720
	НДРЖИЭЗ	HDPXY01
	94	95

agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551: Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Upregulation of CD152 FMAT. CD152 (a.k.a. CTLA-4) expression is restricted to activated T cells. CD152 is a negative regulator of T cell proliferation. Reduced CD152 expression has been linked to hyperproliferative and autoimmune diseases. Overexpression of CD152 may lead to impaired immunoresponses. Assays for immunomodulatory proteins important in the maintenance of T cells homeostasis and expressed almost exclusively on CD4+ and CD8+ T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate the activation of T cells, maintain T cell homeostasis, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers, such as CD152, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J Biomolecular Screening 4.102, 204 (1000). Bouland et al., Il vannbocutes, a practical approach.
	н D QHD03 72
	- 96

HDTBD53	727	10 and activation of T-cells. Myoblast cell proliferation	or routinely modified to assess the ability of polypeptides of the invention (including antibodics and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al. "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "Th-16 per type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirely. Exemplary cells that may be used according to these assays include Th2 cells. IL0 secreted from Th2 cells may be used according to these assays include Th2 cells. IL0 secreted from Th2 cells may be used according to these assays include Th3 cells. Th3 eachs of Th2 cells are secrete IL4. IL0, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells pay a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood. Assays for muscle cell proliferation are well known in the art and may be used or routinely modified Assays for muscle cell proliferation are well known in the art and may be used or routinely modified to test activity of polypeptides and antibodies of the invention) (including agonists or antagonists of the invention) include, for myogenic cells in regenerating skeletal muscles of rats. Dev Growth Differ Apr;43(2):155-64 (2001); Ewton DZ, et al., "IGF binding proteins 4.5 and -6 may play specialized roles during from myogenic cells in regenerating growth factor beta on proliferation of L6 and embryonic portion myo	to the state of th
	}	MCP-1	and act to induce chemotaxis and activation of monocytes and T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce chemotaxis, and modulate immune cell activation. Exemplary assays that test for	7
	HDTBD53		722 723	10 and activation of T-cells. 722 Myoblast cell proliferation 723 Production of MCP-1

immunomodulatory proteins evaluate the production of cell surface markers, such as monocyte chemoattractant protein (MCP), and the activation of monocytes and T cells. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or	otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.		Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
		Production of IL-6	Production of ICAM-1
		723	723
		HDTBP04	HDTBP04
		86	86

				antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or frontinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P. et al.
				FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000),
				the contents of each of which is herein incorporated by reference in its entirety. Cells that may be
				used according to these assays are publicly available (e.g., through the ATCC) and/or may be
				routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
66	HDTBV77	724	Regulation of	Assays for the regulation of transcription through the DMEF1 response element are well-known in
			transcription via	the art and may be used or routinely modified to assess the ability of polypeptides of the invention
			DMEF1 response	(including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response
			element in	element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin
			adipocytes and	production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2
			pre-adipocytes	transcription factor and another transcription factor that is required for insulin regulation of Glut4
				expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat
				and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1
				response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai,
				M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 275(21):16323-8
				(2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair
				regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in
				transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988);
	-			and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is
				herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used
				according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
	-			generated. Exemplary cells that may be used according to these assays include the mouse 3T3-L1
				cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous
				substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte
				to adipose-like conversion under appropriate differentiation culture conditions.
100	ното рез	725	Endothelial Cell	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or
			Apoptosis	routinely modified to assess the ability of polypeptides of the invention (including antibodies and
				agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Induction
				of apoptosis in endothelial cells supporting the vasculature of tumors is associated with tumor
				regression due to loss of tumor blood supply. Exemplary assays for caspase apoptosis that may be
				used or routinely modified to test capase apoptosis activity of polypeptides of the invention

			(including antibodies and agonists or antagonists of the invention) include the assays disclosed in Lee et al., FEBS Lett 485(2-3): 122-126 (2000): Nor et al., J Vasc Res 37(3): 209-218 (2000): and
			Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are
			herein incorporated by reference in its entirety. Endothelial cells that may be used according to
			these assays are publicly available (e.g., through commercial sources). Exemplary endothelial cells
			that may be used according to these assays include bovine aortic endothelial cells (bAEC), which
			are an example of endothelial cells which line blood vessels and are involved in functions that
			include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell
			extravasation.
ното рез	725	Stimulation of	Assays for measuring calcium flux are well-known in the art and may be used or routinely
		Calcium Flux in	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
		pancreatic beta	antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to
		cells.	measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium
			compared to much higher extracellular calcium. Extracellular factors can cause an influx of
			calcium, leading to activation of calcium responsive signaling pathways and alterations in cell
			functions. Exemplary assays that may be used or routinely modified to measure calcium flux by
			polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
			include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995);Mogami H, el
			al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51
			(1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of
			each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used
			according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
			generated. Exemplary pancreatic cells that may be used according to these assays include HITTIS
			Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells
			transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors.
			The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by
			somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219:
			547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
HDTFE17	726	Activation of	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
		transcription	response element are well-known in the art and may be used or routinely modified to assess the
		through NFAT	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
		response element	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
		in immune cells	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
		(such as natural	element that may be used or routinely modified to test NFAT-response element activity of

			killer cells).	polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);
				Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol
				31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993). the contents of each of which are herein incorporated by
				reference in its entirety. NK cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays
				include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.
102	HDTGC73	727	Production of	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely
			ICAM-1	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or
-				routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al,
				FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000),
				the contents of each of which is herein incorporated by reference in its entirety. Cells that may be
				used according to these assays are publicly available (e.g., through the ATCC) and/or may be
				routinely generated. Exemplary cells that may be used according to these assays include
				microvascular endothelial cells (MVEC).
103	HE2DE47	728	Regulation of	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or
			apoptosis in	routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			pancreatic beta	agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis.
			cells.	Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary
				assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity
				of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et
				al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):687-
				94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:S44-7 (2001); Suk K, et al., J Immunol,
				166(7):4481-9 (2001); Tejedo J, et al., FEBS Lett, 459(2):238-43 (1999); Zhang, S., et al., FEBS
				Lett, 455(3):315-20 (1999); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res
_				37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the
····				contents of each of which are herein incorporated by reference in its entirety. Pancreatic cells that
				may be used according to these assays are publicly available (e.g., through the ATCC) and/or may
				be routinely generated. Exemplary pancreatic cells that may be used according to these assays

				include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a radiation induced transplantable ratislet cell tumor. The cells produce and secrete islet polypeptide
				hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1980 77:3519.
104	HE2EN04	729	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin. Nature 410(6224):37-40 (2001); and Cobb MH. Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions, they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamerhasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" (2 in Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Distruption of fas receptor signaling by nitric oxide in eosinophils" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.
105	HE2FV03	730	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163

				(2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include Aortic Smooth Muscle Cells (AOSMC); such as bovine AOSMC.
106	HE2NV57	731	Activation of T-Cell p38 or JNK Signaling Pathway.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an L-2 dependent suspension-culture cell line with cytotoxic activity.
106	HE2NV57	731	Activation of transcription through API response element in immune cells (such as T-cells).	Assays for the activation of transcription through the API response element are known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API response element that may be used or routinely modified to test API-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays include the CTLL CExemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
106	HE2NV57	731	Activation of transcription through cAMP	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP and regulate

		
CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is a suspension culture of IL-2 dependent cytotoxic T cells.	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary mouse T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the CTLL cell line, which is a suspension culture of IL-2 dependent cytotoxic T cells.	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated
response element in immune cells (such as T-cells).	Activation of transcription through GAS response element in immune cells (such as T-cells).	Activation of transcription through serum response element in immune cells (such as T-cells).
	731	731
	HE2NV57	HE2NV57
	106	106

				her enforcement in its ambients. To all that many he wond according to those annual and military annual alla
				by reference in its clinicity. I cens that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays.
				include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic
				activity,
106	HE2NV57	181	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely
				modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is
				measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is
				upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component
				in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of
				insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and
				agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J,
				47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et
				al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52
				(1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of
				each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used
_				according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
				generated. Exemplary pancreatic cells that may be used according to these assays include HITT15
				Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells
				transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors.
				The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by
				somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219:
				547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
106	HE2NV57	731	Activation of	Assays for the activation of transcription through the CD28 response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through CD28	(including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T
			response element	cells. Exemplary assays for transcription through the CD28 response element that may be used or
			in immune cells	routinely modified to test CD28-response element activity of polypeptides of the invention
			(such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol
	-			159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J
				Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference
		·		in its entirety. T cells that may be used according to these assays are publicly available (e.g.,

				through the ATCC). Exemplary himan T cells that may be used according to these assays include
				the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.
107	нЕ2РН36	732	Regulation of viability and	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including
			proliferation of	antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of
			pancreatic beta	pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the
			coms.	presence of metabolically active cells. Exemplary assays that may be used or routinely modified to
				test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in:
				Friedrichsen Biv, et al., Mol Endocrinol, 13(1):130-48 (2001); Huotari M.A, et al., Endocrinology, 139(4):1494-9 (1998): Hipl SR et al., 18iol Chem 1998 Inl 10:373(28):17771-9 (1998): the
				contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that
				may be used according to these assays are publicly available (e.g., through the ATCC) and/or may
				be routinely generated. Exemplary pancreatic cells that may be used according to these assays
				include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated
				from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of
				native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al.
				Endocrinology 1992 130:16/.
801	HE6EU50	733	Activation of	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			transcription	response element are well-known in the art and may be used or routinely modified to assess the
			through NFAT	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			response in	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			immune cells	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
			such as T-cells).	element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);
		,		Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol
				31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J
				Biol Chem 268(19): 14285-14293 (1993), the contents of each of which are herein incorporated by
				reference in its entirety. T cells that may be used according to these assays are publicly available
				(e.g., unougn the ATCC). Exemplary numan 1 cells that may be used according to these assays

				include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.	
108	HEGEUSO	733	Activation of transcription through GAS response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the MOLT4 cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).	de that T able
108	небеизо	733	Upregulation of CD69 and activation of T cells	CD69 FMAT. CD69 is an activation marker that is expressed on activated T cells, B cells, and NK cells. CD69 is not expressed on resting T cells, B cells, or NK cells. CD69 has been found to be associated with inflammation. Assays for immunomodulatory proteins expressed in T cells, B cells, and leukocytes are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate the activation of T cells, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers, such as CD69, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ferenczi et al., J Autoimmun 14(1):63-78 (200); Werfel et al., Allergy 52(4):465-469 (1997); Taylor-Fishwick and Siegel, Eur J Immunol 25(12):3215-3221 (1995); and Afetra et al., Ann Rheum Dis 52(6):457-460 (1993), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.	ATK (TK Ility In) (In) (In) (In) (In) (In) (In) (In)

			10 and activation	or routinely modified to assess the ability of nolvnentides of the invention (including antibodies and
			of T-cells.	agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation
				of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of
				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
				modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed
	- 11			and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-
				968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology &
				Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
				reference in their entirety. Exemplary cells that may be used according to these assays include Th2
				cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells
				are a class of T cells that secrete L4, L10, L13, L5 and L6. Factors that induce differentiation
				and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and
				asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions
				using peripheral blood lymphocytes isolated from cord blood.
111	HE9HY07	736	Activation of	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that
			Adipocyte ERK	regulate cell proliferation or differentiation are well known in the art and may be used or routinely
			Signaling Pathway	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation.
				Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK
				kinase-induced activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-
				1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999);
				Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40
				(2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of
				which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used
				according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse
				adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an
				adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells
				developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under
				appropriate differentiation conditions known in the art.
111	HE9HY07	736	Regulation of	Assays for the regulation of transcription through the FAS promoter element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through the FAS	(including antibodies and agonists or antagonists of the invention) to activate the FAS promoter
			promoter element	element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis.

FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Biochem J, 317 (Pt 1):257-65 (1996); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays, such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Preadipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
in hepatocytes	Activation of transcription through cAMP response element (CRE) in preadipocytes.	Activation of transcription
	737	737
	HE9NN84	HE9NN84
	112	112

production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 83:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell. This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Tumer et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
through GATA-3 produresponse element well-hin immune cells transc cells). (such as mast develocells). (such as mast develocells). (aschool of the invention of the invention of the invention of the invention of the invention of the included of the incl	Activation of This ranscription transcription line. A
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				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
113	HEBEJ18	738	Activation of T. Cell p38 or JNK Signaling Pathway.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
114	HEEAQ11	739	Regulation of viability or proliferation of immune cells (such as human eosinophil EOL-1 cells).	Assays for the regulation (i.e. increases or decreases) of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of eosinophil cells and cell lines. For example, the CellTiter-Gloô Luminescent Cell Viability Assay (Promega Corp., Madison, WI, USA) can be used to measure the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Eosinophils are a type of immune cell important in allergic responses; they are recruited to tissues and mediate the inflammtory response of late stage allergic reaction. Eosinophil cell lines that may be used according to these assays are publicly available and/or may be routinely generated. Exemplary eosinophil cells that may be used according to these assays include EOL-1 Cells.
114	HEEAQ11	739	Activation of T-Cell p38 or JNK Signaling Pathway.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem

				379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001): and Cobb
				MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are
				publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an L-2 dependent suspension-culture cell line
	, , O , Chara.			with cytotoxic activity.
114	HEEAQII	739	Upregulation of	CD71 FMAT. CD71 is the transferrin receptor. Transferrin is a major iron carrying protein that is
			CD/1 and	essential for cell proliferation. CD/1 is expressed predominantly on cells that are actively
			activation of 1 cells	proliterating. Assays for immunomodulatory proteins expressed on activated 1 cells, B cells, and most proliferating cells are well known in the art and may be used or routinely modified to assess
				the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
				invention) to modulate the activation of T cells, and/or mediate humoral or cell-mediated immunity.
				Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface
				markers, such as CD71, and the activation of T cells. Such assays that may be used or routinely
				modified to test immunomodulatory activity of polypeptides of the invention (including antibodies
				and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia
				et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical
				approach" Chapter 6:138-160 (2000); and Afetra et al., Ann Rheum Dis 52(6):457-460 (1993), the
				contents of each of which are herein incorporated by reference in its entirety. Human T cells that
				may be used according to these assays may be isolated using techniques disclosed herein or
				otherwise known in the art. Human T cells are primary human lymphocytes that mature in the
				thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-
				mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory
115	HEEB105	740	Activation of	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell
			transcription	line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production.
			through NFAT	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			response element	response element are well-known in the art and may be used or routinely modified to assess the
			in immune cells	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
<u> </u>			(such as mast	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
-				element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)

741 Production of ICAM-1 742 Stimulation of insulin secretion from pancreatic beta cells.	
	742 Stimulation of insulin secretion from pancreatic beta cells.
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				typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
118	HEPAB80	743	Activation of Adipocyte ERK Signaling Pathway	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays include 373-L1 cells. 373-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 373 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
118	HEPAB80	743	Regulation of viability and proliferation of pancreatic beta cells.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ohtani KI, et al., Endocrinology, 139(1):172-8 (1998); Krautheim A, et al, Exp Clin Endocrinol Diabetes, 107 (1):29-34 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord

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				and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
119	HEQAK71	744	Production of TNF alpha by dendritic cells	TNFa FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells, fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and cytotoxic effects on a variety of cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, modulate inflammation and
				cytotoxicity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines such as tumor necrosis factor alpha (TNFa), and the induction or inhibition of an inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or anapomists of the invention) include assays disclosed in Miraglia et al. 1 Riomolecular Screening
				4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Verhasselt et al., Eur J Immunol 28(11):3886-3890 (1198); Dahlen et al., J Immunol 160(7):3585-3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety.
				Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.
119	HEQAK71	744	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be
120	HEQCC55	745	Production of MCP-1	microvascular endothelial cells (MVEC). MCP-1 FMAT. Assays for immunomodulatory proteins that are produced by a large variety of cells and act to induce chemotaxis and activation of monocytes and T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce the modulate immuno cell activation. Exemplary assays that test for

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immunomodulatory proteins evaluate the production of cell surface markers, such as monocyte chemoattractant protein (MCP), and the activation of monocytes and T cells. Such assays that may be used or routinely modified to test immunomodulatory and diffferentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science; 282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	This reporter assay measures activation of the NFkB signaling pathway in Ku812 human basophil cell line. Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists)	
	Production of IL-13 and activation of T-cells.	Activation of transcription through NFKB response element in immune cells (such as basophils).	
	745	746	
	HEQCC55	HERAR44	
	120	121	

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or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Marone et al, Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Basophils that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human basophil cell lines that may be used according to these assays include Ku812, originally established from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that can be induced to differentiate into mature basophils.	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al., FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al., FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol,
	Production of ICAM-1	Production of ICAM-1	Stimulation of insulin secretion from pancreatic beta cells.
	746	747	748
	HERAR44	HETBR16	HFABH95
	121	122	123

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				2//(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3/35-40 (1997); Kim, K.H., et al., HRBS 1 et 377(2):237-9 (1995); and Miraglia S et al., Immal of Biomolecular Screening 4:103
				204 (1999), the contents of each of which is herein incorporated by reference in its entirety.
	-	_		Pancreatic cells that may be used according to these assays are publicly available (e.g., through the
				ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according
				to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from
				cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics
				typical of native pancreatic beta cells including glucose inducible insulin secretion. References:
5	20110	9,1		Astari et al. Endocrinology 1992 130:16/.
123	HFABH95	748	Upregulation of	CD69 FMAT. CD69 is an activation marker that is expressed on activated T cells, B cells, and NK
			CD69 and	cells. CD69 is not expressed on resting T cells, B cells, or NK cells. CD69 has been found to be
			activation of T	associated with inflammation. Assays for immunomodulatory proteins expressed in T cells, B cells,
			cells	and leukocytes are well known in the art and may be used or routinely modified to assess the ability
				of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				to modulate the activation of T cells, and/or mediate humoral or cell-mediated immunity.
				Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface
				markers, such as CD69, and the activation of T cells. Such assays that may be used or routinely
		•		modified to test immunomodulatory activity of polypeptides of the invention (including antibodies
				and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia
				et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical
				approach" Chanter 6:138-160 (2000): Ferenczi et al., I Autoimmun 14(1):63-78 (200): Werfel et
				al. Allerov \$2(4):465-469 (1997): Taylor-Fishwick and Siegel Fire I Immunol 25(12):4215-3221
				(1995); and Afetra et al., Ann Rheum Dis 52(6):457-460 (1993), the contents of each of which are
				herein incorporated by reference in its entirety. Human T cells that may be used according to these
				assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T
				cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and
				CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be
				preactivated to enhance responsiveness to immunomodulatory factors.
124	HFAEF57	749	Regulation of	Assays for the regulation of transcription through the FAS promoter element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through the FAS	(including antibodies and agonists or antagonists of the invention) to activate the FAS promoter
			promoter element	element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis.
			in hepatocytes	FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS
_				gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat

				glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Biochem J, 317 (Pt 1):257-65 (1996); Berger, et al., Gene 66:1-10 (1988); and, Cullen B, et al., Biochem J, 317 (Pt 1):257-65 (1996); Berger, et al., Gene 66:1-10 (1988); and, Cullen B, et al., Biochem J, 317 (Pt 1):257-65 (1990); He contact of any of thick in Francisco and agonists of any of the contact of any of the contact of any of the contact of any of the contact of any of the contact of any of the contact of any of the contact of any of the contact of any of the contact
				current, D., et al., predictors in Europinot. 210.302–300 (1972), the contents of each of which is neighborhoopstated by reference in its entirety. Hepatocytes that may be used according to these assays, such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.
125	HFAMB72	750	Activation of JNK Signaling Pathway	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of prolynemides of the invention (including antibodies and aponists or antagonists of the invention) to
			(such as eosinophils).	property of the proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides
				of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999): Kyriakis IM Biochem Soc Symp 64:29-48 (1999): Chang and Karin
				Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that
				may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of
				late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation or apportise in
				essinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-
				activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med;
				Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced
				phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal
				each of which are herein incorporated by reference in its entirety.

126	HFCCQ50	751	Production of	TNFa FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells,
			dendritic cells	cytotoxic effects on a variety of cells are well known in the art and may be used or routinely
				modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, modulate inflammation and
_				cytotoxicity. Exemplary assays that test for immunomodulatory proteins evaluate the production of evaluate the production of an evaluate the production of an evaluate such as tumor necrosis factor alpha (TNFa) and the induction or inhibition of an
				inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test
				immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include assays disclosed in Miraglia et al., J. Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes; a practical approach" Chapter 6:138-160 (2000);
				Verhasselt et al., Eur J Immunol 28(11):3886-3890 (1198); Dahlen et al., J Immunol 160(7):3585-
				3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol
				65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety.
				Human dendritic cells that may be used according to these assays may be isolated using techniques
			_	disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in
				suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell
				proliferation and functional activities.
126	HFCCQ50	751	Production of IL-4	L4 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells that stimulate B cells, T
	٠			cells, macrophages and mast cells and promote polarization of CD4+ cells into TH2 cells are well
				known in the art and may be used or routinely modified to assess the ability of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) to mediate
				immunomodulation, stimulate immune cells, modulate immune cell polarization, and/or mediate
				humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins
				evaluate the production of cytokines, such as IL-4, and the stimulation of immune cells, such as B
				cells, T cells, macrophages and mast cells. Such assays that may be used or routinely modified to
				test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists
		_		or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular
				Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-
	•			160 (2000); Gonzalez et al., J Clin Lab Anal 8(5):277-283 (1194); Yssel et al., Res Immunol
				144(8):610-616 (1993); Bagley et al., Nat Immunol 1(3):257-261 (2000); and van der Graaff et al.,
				Rheumatology (Oxford) 38(3):214-220 (1999), the contents of each of which are herein
				incorporated by reference in its entirety. Human T cells that may be used according to these assays
				may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are

				primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.
126	нъссQ50	751	Activation of transcription through NFKB response element in immune cells (such as the Jurkat human T cell line).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al., Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce L-2 when stimulated.
126	НРССQ50	751	Activation of transcription through GAS response element in immune cells (such as monocytes).	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Gustafson KS, et al., J Biol Chem, 271(33):20035-20046 (1996); Eilers A, et al., Immunobiology, 193(2-4):328-333 (1995); Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Hentinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary immune cells that may be used according to these assays include the I1937 cell line, which is a monocytic cell line.
127	HFCEB37	752	Regulation of	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be

used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipoocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 12(11):1778-91 (1998); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362–368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays includes the H4IIE rat liver hepatoma cell line.	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed inThai, M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays are publicly available (e.g., through the sassays include the mouse 373-1.
transcription of Malic Enzyme in adipocytes	Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes
	753
,	HFFAD59
	128

				cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte
			:	to adipose-like conversion under appropriate differentiation culture conditions.
128	HFFAD59	753	Activation of	Assays for the activation of transcription through the AP1 response element are known in the art and
			transcription	may be used or routinely modified to assess the ability of polypeptides of the invention (including
	·		through AP1	antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions.
			response element	Exemplary assays for transcription through the AP1 response element that may be used or routinely
			in immune cells	modified to test AP1-response element activity of polypeptides of the invention (including
			(such as T-cells).	antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al.,
				Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al.,
				Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811
				(1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol
				29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its
				entirety. T cells that may be used according to these assays are publicly available (e.g., through the
				ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL
				cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
128	HFFAD59	753	Activation of	Assays for the activation of transcription through the Serum Response Element (SRE) are well-
			transcription	known in the art and may be used or routinely modified to assess the ability of polypeptides of the
			through serum	invention (including antibodies and agonists or antagonists of the invention) to regulate the serum
			response element	response factors and modulate the expression of genes involved in growth. Exemplary assays for
			in immune cells	transcription through the SRE that may be used or routinely modified to test SRE activity of the
			(such as T-cells).	polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and
				Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated
				by reference in its entirety. T cells that may be used according to these assays are publicly available
			-	(e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays
	• •			include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic
				activity.
129	HFGAD82	754	Activation of	Assays for the activation of transcription through the API response element are known in the art and
			transcription	may be used or routinely modified to assess the ability of polypeptides of the invention (including
			through AP1	antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions.
			response element	Exemplary assays for transcription through the API response element that may be used or routinely
			in immune cells	modified to test API-response element activity of polypeptides of the invention (including

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antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Mouse T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the HT2 cell line, which is an IL-2 dependent suspension culture cell line that also responds to IL-4.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is antagonists of the invention) to stimulate insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Name 410(824):37-40 (2001); and Cobb MH. Pros Biophys Mol Biol 71(3-4):479-500 (1999); the
(such as T-cells).	Stimulation of insulin secretion from pancreatic beta cells.	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).
	754	755
	HFGAD82	HFIIZ70
	129	130

				contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late
				stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of
				late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to
				assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists
				of the invention) to modulate signal transduction, cell proliteration, activation, or apoptosis in
				eosinophils include assays disclosed and/or cited in: Znang Jr, et al., Kole of caspases in decomplished induced anomical and activation of a lim NED forming binds and a 28 mitores.
				activated protein kinase in human eosinophils" (Jin Exp Immunol: Oct:122(1):20-7 (2000):
				Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med;
				Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR,
				et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced
				phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal
				kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of
				each of which are herein incorporated by reference in its entirety.
131	HFTUR 10	756	Regulation of	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and
			viability and	may be used or routinely modified to assess the ability of polypeptides of the invention (including
			proliferation of	antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of
			pancreatic beta	pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the
			cells.	number of viable cells in culture based on quantitation of the ATP present which signals the
				presence of metabolically active cells. Exemplary assays that may be used or routinely modified to
				test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in:
				Ohtani KI, et al., Endocrinology, 139(1):172-8 (1998); Krautheim A, et al, Exp Clin Endocrinol
				Diabetes, 107 (1):29-34 (1999), the contents of each of which is herein incorporated by reference in
				its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g.,
				through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be
				used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line
				established from Syrian hamster islet cells transformed with SV40. These cells express glucagon,
				somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose
	,			and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord
				and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343,
123	LIEVETIO	757	Domilotion of	1901. Corners Anomicsis Assess for corners anomics are well brown in the ort and may be used or
132	DENET 10	2	Regulation of	Caspase Apopuosis. Assays for caspase apopuosis are well known in the art and may be used of

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routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E-antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., J Exp Med, 192(8):1093-1103 (2000);Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
apoptosis of immune cells (such as mast cells).	Activation of transcription through NFAT response in immune cells (such as T-cells).	Activation of Natural Killer Cell ERK Signaling
		757
	HFKET18	HFKET18
	132	132

			Pathway.	antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for FRK kinase activity that may be used or routinely modified to test FRK
				kinase-induced activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature
				410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the
				contents of each of which are herein incorporated by reference in its entirety. Natural killer cells
				that may be used according to these assays are publicly available (e.g., through the ATCC).
				Exemplary natural killer cells that may be used according to these assays include the human natural
				killer cell lines (for example, NK-YT cells which have cytolytic and cytotoxic activity) or primary NK cells.
133	HFOXA73	758	Production of IL-	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used
			10 and activation	or routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			of T-cells.	agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation
_				of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of
				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
				modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed
				and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-
				968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology &
				Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
				reference in their entirety. Exemplary cells that may be used according to these assays include Th2
				cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells
				are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation
				and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and
-				asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions
		1		using peripheral blood lymphocytes isolated from cord blood.
134	HFPA071	759	Production of IL-8	Assay that measures the production of the chemokine interleukin-8 (IL-8) from immune cells (such
_			by immune cells	as the EOL-1 human eosinophil cell line) are well known in the art (for example, measurement of
			(such as the	L-8 production by FMAT) and may be used or routinely modified to assess the ability of
			human EOL-1	polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to
			eosinophil cells)	promote or inhibit. Eosinophils are a type of immune cell important in allergic responses; they are
				recruited to tissues and mediate the inflammtory response of late stage allergic reaction. IL8 is a
				strong immunomodulator and may have a potential proinflammatory role in immunological diseases
				and disorders (such as allergy and asthma).

	247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to	assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" Clin Exp Immunol; Oct; 122(1):20-7 (2000);	Hebestreif H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	
Activation of JNK Signaling Pathway in immune cells (such as eosinophils).				Production of IL-8 by by endothelial cells (such as Human Umbilical Cord Endothelial Cells).
759				759
HFPAO71				HFPAO71
134				134

				An ancientalist and a second s
135	HEPCX00	760	Production of	as incurreptints, macrophiages, and tympinocytes. TNRs RMAT Account for imminormedial atom metains anothered by activated macrombanes. The allo
)		3	TNF alpha by	fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and
			dendritic cells	cytotoxic effects on a variety of cells are well known in the art and may be used or routinely
				modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to mediate immunomodulation, modulate inflammation and
				cytotoxicity Exemplary assays that test for immunomodulatory proteins evaluate the production of
				cytokines such as tumor necrosis factor alpha (TNFa), and the induction or inhibition of an
				inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test
				immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening
				4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000);
				Verhasselt et al., Eur J Immunol 28(11):3886-3890 (1198); Dahlen et al., J Immunol 160(7):3585-
	•			3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol
				65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety.
				Human dendritic cells that may be used according to these assays may be isolated using techniques
				disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in
				suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell
				proliferation and functional activities.
135	HFPCX09	160	Activation of	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast
			transcription	cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
			through GATA-3	production. Assays for the activation of transcription through the GATA3 response element are
			response element	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
			in immune cells	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
			such as mast	transcription factors and modulate expression of mast cell genes important for immune response
			cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test GATA3-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available

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(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the	peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)	response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to apply the contract of the invention o	invention) to regulate 1974.1 transcription factors and inoquite expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of	polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al. Gene 66-1-10 (1998). Cullen and Malm. Methods in	Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De	Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et	al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by	reference in its entirety. Mast cells that may be used according to these assays are publicly available	(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the UMC 1 cell line, which is an immediate human mast cell line actabilished from the	nictude the ray of a patient with mast cell leukemia, and exhibits many characteristics of immature	mast cells.	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS)	response element are well-known in the art and may be used or routinely modified to assess the	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the	invention) to regulate STAT transcription factors and modulate gene expression involved in a wide	variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polymentides of the	invention (including antibodies and agonists or antagonists of the invention) include assays	disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-	368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al.,	Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the	The morning is a factor of the contraction will be a factor of the contraction of the con
		Activation of transcription through NFAT	in immune cells	cells).									Activation of	transcription	through GAS	response element	In immune cells (such as T-cells).					
		092											761									
		HFPCX09											HFRAN90									
		135											136									

				cells, such as the MOLT4 cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).
136	HFRAN90	761	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al., FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
137	HFTBMS0	762	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek; A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
137	HFTBM50	762	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of

				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate II -10 production and/or T-cell proliferation include for example assays such as disclosed
				and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-
				968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Theorem (20, 187, 106, 2000), the contests of each of which are herein incompared by
				Therapeduces, 66: 187-190 (2000), the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2
				cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells
				are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation
				and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and
				asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
138	HFTDL56	763	Production of	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely
			ICAM-1	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or
				routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al,
				FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000),
				the contents of each of which is herein incorporated by reference in its entirety. Cells that may be
				used according to these assays are publicly available (e.g., through the ATCC) and/or may be
				routinely generated. Exemplary cells that may be used according to these assays include
				microvascular endothelial cells (MVEC).
139	HFTDZ36	764	Protection from	Caspase Apoptosis Rescue. Assays for caspase apoptosis rescue are well known in the art and may
			Endothelial Cell	be used or routinely modified to assess the ability of the polypeptides of the invention (including
			Apoptosis.	antibodies and agonists or antagonists of the invention) to inhibit caspase protease-mediated
				apoptosis. Exemplary assays for caspase apoptosis that may be used or routinely modified to test
				caspase apoptosis rescue of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include the assays disclosed in Romeo et al., Cardiovasc Res 45(3):
				788-794 (2000); Messmer et al., Br J Pharmacol 127(7): 1633-1640 (1999); and J Atheroscler
				Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its
				entirety. Endothelial cells that may be used according to these assays are publicly available (e.g.,
				through commercial sources). Exemplary endothelial cells that may be used according to these
				assays include bovine aortic endothelial cells (bAEC), which are an example of endothelial cells
				which line blood vessels and are involved in functions that include, but are not limited to,
				angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.
139	HFTDZ36	764	Stimulation of	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely

			insulin secretion	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
			Irom pancreauc	antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is
			peta cells.	measured by FMA1 using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is
				upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component
				in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of
				insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and
				agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol,
				277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al.,
				FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-
				204 (1999), the contents of each of which is herein incorporated by reference in its entirety.
				Pancreatic cells that may be used according to these assays are publicly available (e.g., through the
				ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according
				to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from
				cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics
				typical of native pancreatic beta cells including glucose inducible insulin secretion. References:
				Asfari et al. Endocrinology 1992 130:167.
140	HFVIC62	765	Activation of	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast
			transcription	cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
			through GATA-3	production. Assays for the activation of transcription through the GATA3 response element are
			response element	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
			in immune cells	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
			(such as mast	transcription factors and modulate expression of mast cell genes important for immune response
			cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test GATA3-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature

140	HFVIC62	765	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in invention) to regulate NFAT transcription factors and modulate expression of genes involved in invention) to regulate NFAT transcription factors and modulate expression of genes involved in include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by the contents of each of which are herein incorporated by the contents of each of which are herein incorporated by the contents of each of which are herein incorporated by the contents of each of which are herein incorporated by the contents of each of which are herein incorporated by the contents of each of which are herein incorporated by the contents of each of which are herein incorporated by the contents of each of which are herein incorporated by the contents of each of which are herein incorporated by the contents of each of which are herein incorporated by the contents of each of which are herein incorporated by the contents of each of which each of which each of the contents of each of each of each of each of each of each of each of each of each of each of each of each of each of each of each each
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
141	HFXAM76	766	Production of GM-CSF	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage. Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory cytokine. Assays for imminomodulatory proteins that momote the production of GM-CSF are well
				known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and modulate the growth and differentiation of leukocytes. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF, and the activation of T cells. Such assays that may be used or routinely modified to test
				immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999), Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-

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160 (2000); and Ye et al., J Leukoc Biol (58(2):225-233, the contents of each of which are herein incorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclosed herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor cells and also recognize antibody bound on target cells, via NK Fc receptors, leading to cell-	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed inThai, MV., et al., J Biol Chem, 273(23): 14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23): 14285-92 (1998); Mora, S., et al., Biol Chem, 273(23): 14285-92 (1998); Mora and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4:275(31):2366-73: Berger, et al., Gene 66:1-10 (1998); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays include the mouse 3T3-L1 cells are a continuous substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al,
160 (2000); and Ye et al., J Leukoc Biol (58(2):225-233, the contents of each of which are herein incorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclo herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes the have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumo cells and also recognize antibody bound on target cells, via NK Fc receptors, leading to cell-	Assays for the regulation of transcription through the DMEF1 response element are well-known in Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insupproduction. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF1 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal musche. GLUT4 is the primary insulin-responsive glucose transporter in fal and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed inTha M.V., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem, 2000 Aug 4:275(31):2366-73; Berger, et al., Gene 66:1-10 (1988 and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays include the mouse 3T3-L. cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipoct to adipose-like conversion under appropriate differentiation culture conditions.	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or roun modified to assess the ability of polypeptides of the invention (including antibodies and agonist antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be us routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al,
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	Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes	Production of ICAM-1
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				used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
144	HFXJU68	692	Activation of transcription through cAMP	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate
			response element (CRE) in pre-	CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate
			adipocytes.	the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adinocytes. CRF contains the binding sequence for the transcription factor.
				CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response
				element that may be used or routinely modified to test cAML-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al. Mol Cell Biol 20(3):1008-1020 (2000): and Klemm et al. I Biol Chem 273:017-023
				(1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-
				adipocytes that may be used according to these assays are publicly available (e.g., through the
				ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used
				according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line
				that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo
				a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
144	HFXJU68	692	Production of IL-	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used
			10 and activation	or routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			of T-cells.	agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation
				of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of
				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
				modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed
				and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-
				968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology &
				Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
				reference in their entirety. Exemplary cells that may be used according to these assays include Th2
				cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells

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are a class of T cells that secrete II 4 II 10 II 13 II 5 and II 6 Hactors that induce differentiation	and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and	asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or	antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is	measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is	upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component	in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of	insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and	agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol,	277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al.,	FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-	204 (1999), the contents of each of which is herein incorporated by reference in its entirety.	Pancreatic cells that may be used according to these assays are publicly available (e.g., through the	ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according	to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from	cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics	typical of native pancreatic beta cells including glucose inducible insulin secretion. References:	Asfari et al. Endocrinology 1992 130:167.	Assays for muscle cell proliferation are well known in the art and may be used or routinely modified	to assess the ability of polypeptides of the invention (including antibodies and agonists or	antagonists of the invention) to stimulate or inhibit myoblast cell proliferation. Exemplary assays	for myoblast cell proliferation that may be used or routinely modified to test activity of polypeptides	and antibodies of the invention (including agonists or antagonists of the invention) include, for	example, assays disclosed in: Soeta, C., et al. "Possible role for the c-ski gene in the proliferation of	myogenic cells in regenerating skeletal muscles of rats" Dev Growth Differ Apr;43(2):155-64	(2001); Ewton DZ, et al., "IGF binding proteins-4, -5 and -6 may play specialized roles during L6	myoblast proliferation and differentiation" J Endocrinol Mar;144(3):539-53 (1995); and, Pampusch	MS, et al., "Effect of transforming growth factor beta on proliferation of L6 and embryonic porcine	myogenic cells" J Cell Physiol Jun; 143(3):524-8 (1990); the contents of each of which are herein	incorporated by reference in their entirety. Exemplary myoblast cells that may be used according to
S		<u> </u>	Stimulation of insulin secretion	reatic					-	-		-	1	7	<u>+</u>	-	-	7	-	proliferation t		-		•			-			
		· - ·	170																771											
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				myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuse to form multinucleated myotubes and striated fibers after culture in differentiation media.
147	HGBFO79	277	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include Aortic Smooth Muscle Cells (AOSMC); such as bovine AOSMC.
147	HGBFO79	772	Proliferation of immune cells (such as the HMC-1 human mast cell line)	Assays for the regulation (i.e. increases or decreases) of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of eosinophil cells and cell lines. For example, the CellTiter-Gloô Luminescent Cell Viability Assay (Promega Corp., Madison, WI, USA) can be used to measure the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Mast cells are found in connective and mucosal tissues throughout the body. Mast cell activation (via immunoglobulin E -antigen, promoted by T helper cell type 2 cytokines) is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Mast cell lines that may be used according to these assays are publicly available and/or may be routinely generated. Exemplary mast cells that may be used according to these assays include HMC-1, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
148	HGBHI35	773	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol,

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277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Bur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary rat natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC).	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., I Immunol 155(10):4582-4587 (1995), the
	Activation of transcription through STAT6 response element in immune cells (such as natural killer cells).	Activation of transcription through GAS response element in immune cells (such as T-cells).
	774	774
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				contents of each of which are herein incorporated by reference in its entirety. Exemplary human T
				cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).
149	HGBIB74	774	Activation of transcription	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the
			through NFAT	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			response element in immune cells	invention) to regulate NFA1 transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response
			(such as natural	element that may be used or routinely modified to test NFAT-response element activity of
			killer cells).	polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);
				Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol
				31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J
				Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by
		_		reference in its entirety. NK cells that may be used according to these assays are publicly available
				(e.g., infough the ATCC). Exemplary numan INK cells that may be used according to these assays
				include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic
149	HGBIB74	774	Activation of	Assays for the activation of transcription through the Serum Response Flement (SRE) are well-
			transcription	known in the art and may be used or routinely modified to assess the ability of polypeptides of the
			through serum	invention (including antibodies and agonists or antagonists of the invention) to regulate serum
			response element	response factors and modulate the expression of genes involved in growth and upregulate the
			in immune cells	function of growth-related genes in many cell types. Exemplary assays for transcription through the
			(such as natural	SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention
			killer cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol
				153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each
				of which are herein incorporated by reference in its entirety. T cells that may be used according to
				these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used
				according to these assays include the NK-YT cell line, which is a human natural killer cell line with
				cytolytic and cytotoxic activity.
150	HGLAF75	775	Regulation of	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be

	
used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays includes the mouse 373-L1 cell line. 373-L1 is a mouse preadipocyte cell line (adherent). It is a continuous substrain of 373 fibroblasts developed through clonal isolation. Cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al.
transcription of Malic Enzyme in hepatocytes	Regulation of viability and proliferation of pancreatic beta cells.
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	HGLAF75
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				Endocrinology 1992 130:167.
150	HGLAF75	775	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely
				modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is
			•	measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is
				upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component
				in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of
				insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and
				agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J,
				47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et
				al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52
				(1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of
				each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used
				according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
				generated. Exemplary pancreatic cells that may be used according to these assays include HITT15
				Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells
				transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors.
				The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by
				somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219:
				547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
151	HHEMA75	9//	Activation of	Assays for the activation of transcription through the cAMP response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through cAMP	(including antibodies and agonists or antagonists of the invention) to increase cAMP, bind to CREB
			response element	transcription factor, and modulate expression of genes involved in a wide variety of cell functions.
			in immune cells	Exemplary assays for transcription through the cAMP response element that may be used or
			(such as T-cells).	routinely modified to test cAMP-response element activity of polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-
				117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which
				are herein incorporated by reference in its entirety. T cells that may be used according to these
				assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used
				according to these assays include the JURKAT cell line, which is a suspension culture of leukemia
				cells that produce IL-2 when stimulated.

Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., I Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays
 Berger et al., Gene 66:1-10 (1998); C Henthorn et al., Proc Natl Acad Sci L 159(3):1319-1327 (1997); Parra et al Biol Chem 3(1):552-560 (1998), the in its entirety. T cells that may be uss through the ATCC). Exemplary hum the SUPT cell line, which is a suspen	Activation of Assays for the activation of transcript transcription response element are well-known in through GAS ability of polypeptides of the inventic invention) to regulate STAT transcript variety of cell functions. Exemplary wariety of cell functions. Exemplary may be used or routinely modified to invention (including antibodies and a disclosed in Berger et al., Gene 66:1-368 (1992); Henthorn et al., Proc Nat Blood 93(6):1980-1991 (1999); and Blood 93(6):1980-1991 (1999); and the Contents of each of which are herein cells, such as the SUPT cell line, that (e.g., through the ATCC).	Activation of response element are well-known in through NFAT ability of polypeptides of the inventic invention) to regulate NFAT transcrip in immune cells information in that may be used or routinely polypeptides of the invention (includ include assays disclosed in Berger et Enzymol 216:362-368 (1992); Henth Serfling et al., Biochim Biophys Acts 31(10):1221-1236 (1999); Fraser et a Biol Chem 268(19):14285-14293 (19 reference in its entirety. T cells that 1 (e.g., through the ATCC). Exemplar
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				include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
151	HHEMA75	776	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-84 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
151	HHEMA75	776	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med J82(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NR cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NR cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.
152	HHENK42	777	Production of IL- 13 and activation of T-cells.	Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test

So or antagonists of the Grunig, G, et al., cience;282: 2261-2263 rgic asthma" Science; 282: by reference in their lude Th2 cells. IL.13, a er-responsiveness and 13, IL.5 and IL.6. Factors in the initiation and ed in in vitro culture under om cord blood.	ent are well-known in the lides of the invention atte the FAS promoter enzyme for lipogenesis. P. Insulin increases FAS tion is also somewhat filed to test for FAS on (including antibodies flong, S., et al., Proc Natl 260(3):743-51 (1999); hene 66:1-10 (1988); and, of each of which is herein scording to these assays, or may be routinely isays include rat liver rivatives.	nent are well-known in the ides of the invention ease cAMP, regulate a wide variety of cell ntify factors that activate and is involved in
activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science;282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL 10, IL 13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Assays for the regulation of transcription through the FAS promoter element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis. FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Biochem J, 317 (Pt 1):257-65 (1996); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays, such as HAIIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in
activity of polypeptides and invention) include, for examinention IL-13 inde (1998), and Wills-Karp M, 6 2258-2261 (1998); the contentiety. Exemplary cells the Th2 type cytokine, is a pote allergic asthma. Th2 cells at that induce differentiation at pathogenesis of allergy and Th2 polarizing conditions us	Assays for the regulation of art and may be used or routi (including antibodies and ag element in a reporter construction in a reporter construction in livers or glucose dependent. Exemply promoter element activity (in and agonists or antagonists of Acad Sci U.S.A., 97(8):394). Oskouian B, et al., Methods in incorporated by reference in such as H4IIE cells, are pub generated. Exemplary hepat pepatoma cell line(s) inducitional.	Assays for the activation of art and may be used or routi (including antibodies and ag CREB transcription factors, functions. For example, a 3 the cAMP signaling pathwa;
	Regulation of transcription through the FAS promoter element in hepatocytes	Activation of transcription through cAMP response element (CRE) in preadipocytes.
	778	779
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·	153	154

				CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response
_				element that may be used or routinely modified to test CAMLY-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
		_		Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al. Mol Cell Riol 20(3):1008-1020 (2000): and Klemm et al. 1 Riol Chem 273:017-023
		_ 		(1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-
				adipocytes that may be used according to these assays are publicly available (e.g., through the
				ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used
	•			according to these assays include 313-E1 cells. 313-E1 is an adject mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo
				a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the
				art.
154	HHEPM33	779	Activation of	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast
			transcription	cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
			through GATA-3	production. Assays for the activation of transcription through the GATA3 response element are
			response element	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
		_	in immune cells	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
			(such as mast	transcription factors and modulate expression of mast cell genes important for immune response
			cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test GATA3-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
			-	peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
				mast cells.
154	HIHEPM33	779	Activation of transcription	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to extokine and chemokine production.

Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription farctors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary rat natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC).	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the
through NFAT response element in immune cells (such as mast cells).	Activation of transcription through STAT6 response element in immune cells (such as natural killer cells).	Activation of transcription
	779	611
	ННЕРМ33	HHEPM33
	154	154

ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human natural killer cell line with cytolytic and cytotoxic activity.	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in
through GAS response element in immune cells (such as T-cells).	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Activation of transcription through serum response element in immune cells (such as natural killer cells)
	779	779
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	154	154

				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988): Benson et al., I Imminol
				153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each
				of which are herein incorporated by reference in its entirety. T cells that may be used according to
				these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used
				according to these assays include the NK-YT cell line, which is a human natural killer cell line with
155	HHFHJ59	780	Activation of	Assays for the activation of transcription through the cAMP response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through cAMP	(including antibodies and agonists or antagonists of the invention) to increase cAMP and regulate
<u>-</u>			response element	CREB transcription factors, and modulate expression of genes involved in a wide variety of cell
			in immune cells	functions. Exemplary assays for transcription through the cAMP response element that may be used
			(such as T-cells).	or routinely modified to test cAMP-response element activity of polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-
				117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which
				are herein incorporated by reference in its entirety. T cells that may be used according to these
				assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used
				according to these assays include the CTLL cell line, which is a suspension culture of IL-2
				dependent cytotoxic T cells.
155	HHFHI59	780	Upregulation of	HLA-DR FMAT. MHC class II is essential for correct presentation of antigen to CD4+ T cells.
			HLA-DR and	Deregulation of MHC class II has been associated with autoimmune diseases (e.g., diabetes,
			activation of T	rheumatoid arthritis, systemic lupus erythematosis, and multiple sclerosis). Assays for
			cells	immunomodulatory proteins expressed on MHC class II expressing T cells and antigen presenting
				cells are well known in the art and may be used or routinely modified to assess the ability of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to
				modulate the activation of T cells, and/or mediate humoral or cell-mediated immunity. Exemplary
				assays that test for immunomodulatory proteins evaluate the upregulation of MHC class II products,
				such as HLA-DR antigens, and the activation of T cells. Such assays that may be used or routinely
				modified to test immunomodulatory activity of polypeptides of the invention (including antibodies
				and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia
				et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical
				approach" Chapter 6:138-160 (2000); Lamour et al., Clin Exp Immunol 89(2):217-222 (1992);

				Hurme and Sihvola, Immunol Lett 20(3):217-222 (1989); Gansbacher and Zier, Cell Immunol 117(1):22-34 (1988); and Itoh et al., J Histochem Cytochem 40(11):1675-1683, the contents of each
				of which are herein incorporated by reference in its entirety. Human T cells that may be used
			-	according to these assays may be isolated using techniques disclosed herein or otherwise known in
				the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T
				Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and
				may be preactivated to enhance responsiveness to immunomodulatory factors.
155	HHFHJ59	780	Upregulation of	CD71 FMAT. CD71 is the transferrin receptor. Transferrin is a major iron carrying protein that is
			CD71 and	essential for cell proliferation. CD71 is expressed predominantly on cells that are actively
			activation of T	proliferating. Assays for immunomodulatory proteins expressed on activated T cells, B cells, and
			cells	most proliferating cells are well known in the art and may be used or routinely modified to assess
				the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
				invention) to modulate the activation of T cells, and/or mediate humoral or cell-mediated immunity.
				Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface
				markers, such as CD71, and the activation of T cells. Such assays that may be used or routinely
				modified to test immunomodulatory activity of polypeptides of the invention (including antibodies
	•			and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia
				et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical
				approach" Chapter 6:138-160;(2000); and Afetra et al., Ann Rheum Dis 52(6):457-460 (1993), the
				contents of each of which are herein incorporated by reference in its entirety. Human T cells that
				may be used according to these assays may be isolated using techniques disclosed herein or
				otherwise known in the art. Human T cells are primary human lymphocytes that mature in the
				thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-
				mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory
		1		tactors.
155	HHFHJ59	780	Upregulation of	CD69 FMAT. CD69 is an activation marker that is expressed on activated T cells, B cells, and NK
			CD69 and	cells. CD69 is not expressed on resting T cells, B cells, or NK cells. CD69 has been found to be
			activation of T	associated with inflammation. Assays for immunomodulatory proteins expressed in T cells, B cells,
			cells	and leukocytes are well known in the art and may be used or routinely modified to assess the ability
				of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				to modulate the activation of T cells, and/or mediate humoral or cell-mediated immunity.
				Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface
				markers, such as CD69, and the activation of T cells. Such assays that may be used or routinely
				modified to test immunomodulatory activity of polypeptides of the invention (including antibodies

				FEBS Lett, 31/(2):23/-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incompared by reference in its entirety
				Pancreatic cells that may be used according to these assays are publicly available (e.g., through the
				ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according
				to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from
				cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics
				typical of native pancreatic beta cells including glucose inducible insulin secretion. References:
157	707000111	202	0.1	Astarr et al. Endocrinology 1992 130:167.
/CI	HHGCM/6	78/	Stimulation of	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely
			insulin secretion	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
			from pancreatic	antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is
			beta cells.	measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is
				upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component
				in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of
				insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and
				agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol.
				277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al.,
				FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-
				204 (1999), the contents of each of which is herein incorporated by reference in its entirety.
				Pancreatic cells that may be used according to these assays are publicly available (e.g., through the
				ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according
				to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from
		•		cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics
				typical of native pancreatic beta cells including glucose inducible insulin secretion. References:
				Asfari et al. Endocrinology 1992 130:167.
157	HHGCM76	782	Production of	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely
			ICAM-1	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or
				routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al,
				FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000),
				the contents of each of which is herein incorporated by reference in its entirety. Cells that may be
				used according to these assays are publicly available (e.g., through the ATCC) and/or may be
				routinely generated. Exemplary cells that may be used according to these assays include
				microvascular endothelial cells (MVEC).

Assays for the activation of transcription through the API response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API response element that may be used or routinely modified to test API-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL 10, IL 13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed
Activation of transcription through AP1 response element in immune cells (such as T-cells).	Production of IL- 10 and activation of T-cells.	Production of IL- 10 and activation of T-cells.
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HHGDW43	HHGDW43	ниресо
158	158	159

and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL 10, IL 13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995);Mogami H, et al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
·	Stimulation of Calcium Flux in pancreatic beta cells.	Activation of transcription through serum response element in immune cells (such as T-cells).
	785	786
	HHPEN62	HHSDX28
	160	161

include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.	TNFa FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells, fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and cytotoxic effects on a variety of cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulatory proteins evaluate the production of cytotoxicity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines such as tumor necrosis factor alpha (TNFa), and the induction or inhibition of an inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Verhasselt et al., Eur J Immunol 128(11):386-3890 (1198); Dahlen et al., J Immunol 160(7):3585-3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the
	Production of TNF alpha by dendritic cells	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).
	786	786
	HHSDX28	HHSDX28
	161	161

		
disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J. 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999); He contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs. Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
	Insulin Secretion	Production of IL- 10 and activation of T-cells.
	787	788
	HJABB94	HJABX32
·	162	163

				modulate IL-10 production and/or Tcell proliferation include, for example, assays such as disclosed
				and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology &
				Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
				reference in their entirety. Exemplary cells that may be used according to these assays include Th2
		_		are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation
		_		and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and
				asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using neripheral blood lymphocytes isolated from cord blood
164	HJACG30	789	Activation of	Assays for the activation of transcription through the Serum Response Element (SRE) are well-
		_	transcription	known in the art and may be used or routinely modified to assess the ability of polypeptides of the
			through serum	invention (including antibodies and agonists or antagonists of the invention) to regulate the serum
			response element	response factors and modulate the expression of genes involved in growth. Exemplary assays for
			in immune cells	transcription through the SRE that may be used or routinely modified to test SRE activity of the
			(such as T-cells).	polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and
				Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated
				by reference in its entirety. T cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays
				include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic
				activity.
16 4	HJACG30	789	Stimulation of	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely
			insulin secretion	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
		-	from pancreatic	antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is
			beta cells.	measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is
				upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component
				in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of
				insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and
				agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol,
				277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al.,
				FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-
				204 (1999), the contents of each of which is herein incorporated by reference in its entirety.

				December 2 all above the read according to above account on militals (the rest the
				ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according
				to these assays include rat INN-1 cells. INN-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics
				typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
165	HJBCY35	790	Regulation of	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and
			viability and	may be used or routinely modified to assess the ability of polypeptides of the invention (including
			proliferation of	antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of
			pancreatic beta	pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the
			cells.	number of viable cells in culture based on quantitation of the ATP present which signals the
				presence of metabolically active cells. Exemplary assays that may be used or routinely modified to
				test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in:
				Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology,
				139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the
				contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that
				may be used according to these assays are publicly available (e.g., through the ATCC) and/or may
				be routinely generated. Exemplary pancreatic cells that may be used according to these assays
				include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated
				from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of
				native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al.
				Endocrinology 1992 130:167.
165	HJBCY35	790	Activation of	Kinase assay. Kinase assays, for example an GSK-3 kinase assay, for PI3 kinase signal
			Skeletal Mucle	transduction that regulate glucose metabolism and cell survivial are well-known in the art and may
			Cell PI3 Kinase	be used or routinely modified to assess the ability of polypeptides of the invention (including
			Signalling	antibodies and agonists or antagonists of the invention) to promote or inhibit glucose metabolism
			Pathway	and cell survival. Exemplary assays for PI3 kinase activity that may be used or routinely modified
				to test PI3 kinase-induced activity of polypeptides of the invention (including antibodies and
				agonists or antagonists of the invention) include assays disclosed in Forrer et al., Biol Chem 379(8-
				9):1101-1110 (1998); Nikoulina et al., Diabetes 49(2):263-271 (2000); and Schreyer et al., Diabetes
				48(8):1662-1666 (1999), the contents of each of which are herein incorporated by reference in its
				entirety. Rat myoblast cells that may be used according to these assays are publicly available (e.g.,
				through the ATCC). Exemplary rat myoblast cells that may be used according to these assays

				include L6 cells. L6 is an adherent rat myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuses to form multinucleated myotubes and striated fibers after culture in differentiation media.
166	HJMBN89	791	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2:cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
167	HJPAD75	792	Activation of T-Cell p38 or JNK Signaling Pathway.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
167	HJPAD75	792	Production of IL-6	L-6 FMAT. L-6 is produced by T cells and has strong effects on B cells. L-6 participates in L-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). L-6

	
induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and differentiation and modulate T cell proliferation and function. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and functional activities. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4: 193-204(1999); Rowland et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and unregulate T cell proliferation and functional activities.	Assays for the regulation of transcription through the FAS promoter element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis. FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Methods in Enzymol. 216:362–368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays, such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.
	Regulation of transcription through the FAS promoter element in hepatocytes
	792
	HIPAD75
	167

Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Induction of apoptosis in endothelial cells supporting the vasculature of tumors is associated with tumor regression due to loss of tumor blood supply. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays include bovine aortic endothelial cells (bAEC), which are an example of endothelial cells which line blood vessels and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription
Endothelial Cell Apoptosis	Activation of transcription through NFAT response in immune cells (such as T-cells).	Activation of transcription through NFKB
793	793	793
HKAB184	HKAB184	HKABI84
168	168	168

			response element in immune cells	factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-
			(such as T-cells).	response element activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen
	-			and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA
				85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-
				844 (1999), the contents of each of which are herein incorporated by reference in its entirety.
				Exemplary human T cells, such as the MOLT4, that may be used according to these assays are
				publicly available (e.g., through the ATCC).
169	HKABZ65	794	Production of IL-6	L-6 FMAT. L-6 is produced by T cells and has strong effects on B cells. L-6 participates in L-4
				induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6
				induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease,
				plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for
				immunomodulatory and differentiation factor proteins produced by a large variety of cells where the
				expression level is strongly regulated by cytokines, growth factors, and hormones are well known in
			-	the art and may be used or routinely modified to assess the ability of polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) to mediate immunomodulation
				and differentiation and modulate T cell proliferation and function. Exemplary assays that test for
				immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation
				and upregulation of T cell proliferation and functional activities. Such assays that may be used or
				routinely modified to test immunomodulatory and diffferentiation activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
	-			disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al.,
				"Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Verhasselt et al., J Immunol
				158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its
				entirety. Human dendritic cells that may be used according to these assays may be isolated using
				techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen
_				presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate
				and upregulate T cell proliferation and functional activities.
169	HKABZ65	794	Activation of	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation,
			Endothelial Cell	activation, or apoptosis are well known in the art and may be used or routinely modified to assess
			p38 or JNK	the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			Signaling	invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for
			Pathway.	JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-

			·	induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998), Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48
				(1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its
				entirety. Endothelial cells that may be used according to these assays are publicly available (e.g.,
				through the ATCC). Exemplary endothelial cells that may be used according to these assays include
				human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous
-				blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.
169	HKABZ65	794	Regulation of	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or
			apoptosis in	routinely modified to assess the ability of polypeptides of the invention (including antibodies and
_			pancreatic beta	agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis.
			cells.	Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary
				assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity
				of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et
				al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):687-
				94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:S44-7 (2001); Suk K, et al., J Immunol,
				166(7):4481-9 (2001); Tejedo J, et al., FEBS Lett, 459(2):238-43 (1999); Zhang, S., et al., FEBS
				Lett, 455(3):315-20 (1999); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res
				37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the
				contents of each of which are herein incorporated by reference in its entirety. Pancreatic cells that
				may be used according to these assays are publicly available (e.g., through the ATCC) and/or may
				be routinely generated. Exemplary pancreatic cells that may be used according to these assays
				include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a
				radiation induced transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide
				hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et
				al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1980 77:3519.
170	HKACB56	795	Myoblast cell	Assays for muscle cell proliferation are well known in the art and may be used or routinely modified
			proliferation	to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to stimulate or inhibit myoblast cell proliferation. Exemplary assays
				for myoblast cell proliferation that may be used or routinely modified to test activity of polypeptides
				and antibodies of the invention (including agonists or antagonists of the invention) include, for

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(2001); Ewton DZ, et al., "IGF binding proteins-4, -5 and -6 may play specialized roles during L6 myoblast proliferation and differentiation" J Endocrinol Mar,144(3):539-53 (1995); and, Pampusch
MS, et al., "Effect of transforming growth factor beta on proliferation of L6 and embryonic porcine myogenic cells" J Cell Physiol Jun;143(3):524-8 (1990); the contents of each of which are herein
incorporated by reference in their entirety. Exemplary myoblast cells that may be used according to
misse assays include the fat higherest to cent fille. Nat higherest to cens are an adjicted myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuse to form
multinucleated myotubes and striated fibers after culture in differentiation media.
IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils,
and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization
of CD4+ Coils into 1112 cells are well known in the artain may be used of fourthery induffied to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists
of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig
production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity.
Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines,
such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that
may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays
disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al.,
"Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., Blood 92(9):3338-
3345 (1998); Jung et al., Eur J Immunol 25(8):2413-2416 (1995); Mori et al., J Allergy Clin
Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents
of each of which are herein incorporated by reference in its entirety. Human T cells that may be
used according to these assays may be isolated using techniques disclosed herein or otherwise
known in the art. Human T cells are primary human lymphocytes that mature in the thymus and
express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated
immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.
Assays for measuring expression of VCAM are well-known in the art and may be used or routinely
modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to
meaure the upregulation of cell surface VCAM-1 expresssion in endothelial cells. Endothelial cells
are cells that line blood vessels, and are involved in functions that include, but are not limited to,

			andothelial cells	ancionenesis vascular nermeability vascular tone and immine cell extravasation. Evennlary
			(HUVEC))	_ G ≥
				VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors, and contributes to the extravasation of lymphocytes lengocytes and other immine cells from blood
				vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.
170	HKACB56	795	Activation of	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation,
			Endothelial Cell	activation, or apoptosis are well known in the art and may be used or routinely modified to assess
			p38 or JNK	the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			Signaling	invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for
			Pathway.	JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-
				induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of
				the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998);
				Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48
				(1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol
				71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its
				entirety. Endothelial cells that may be used according to these assays are publicly available (e.g.,
				through the ATCC). Exemplary endothelial cells that may be used according to these assays include
				human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous
				blood vessels, and are involved in functions that include, but are not limited to, angiogenesis,
				vascular permeability, vascular tone, and immune cell extravasation.
170	HKACB56	795	Upregulation of	CD152 FMAT. CD152 (a.k.a. CTLA-4) expression is restricted to activated T cells. CD152 is a
			CD152 and	negative regulator of T cell proliferation. Reduced CD152 expression has been linked to
			activation of T	hyperproliferative and autoimmune diseases. Overexpression of CD152 may lead to impaired
			cells	immunoresponses. Assays for immunomodulatory proteins important in the maintenance of T cell
				homeostasis and expressed almost exclusively on CD4+ and CD8+ T cells are well known in the art
				and may be used or routinely modified to assess the ability of polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) to modulate the activation of T
				cells, maintain T cell homeostasis, and/or mediate humoral or cell-mediated immunity. Exemplary
				assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers,
				such as CD152, and the activation of T cells. Such assays that may be used or routinely modified to
				test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists
				or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J
				Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach"

				Chapter 6:138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Curr Opin Immunol 11(3):294-300 (1999); and Saito T, Curr Opin Immunol 10(3):313-321 (1998),
				the contents of each of which are herein incorporated by reference in its entirety. Human T cells
				that may be used according to these assays may be isolated using techniques disclosed herein or
		_		otherwise known in the art. Human T cells are primary human lymphocytes that mature in the
				thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-
				mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory
171	HKACD58	796	Regulation of	Assays for the regulation of transcription through the DMEF1 response element are well-known in
			transcription via	the art and may be used or routinely modified to assess the ability of polypeptides of the invention
			DMEF1 response	(including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response
			element in	element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin
		_	adipocytes and	production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2
			pre-adipocytes	transcription factor and another transcription factor that is required for insulin regulation of Glut4
				expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat
				and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1
	•			response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai,
				M.V., et al., J Biol Chem, 273(23): 14285-92 (1998); Mora, S., et al., J Biol Chem, 275(21): 16323-8
				(2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair
				regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in
				transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988);
		_		and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is
				herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used
				according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
				generated. Exemplary cells that may be used according to these assays include the mouse 3T3-L1
				cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous
				substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte
				to adipose-like conversion under appropriate differentiation culture conditions.
171	HKACD58	962	Activation of	Assays for the activation of transcription through the Serum Response Element (SRE) are well-
			transcription	known in the art and may be used or routinely modified to assess the ability of polypeptides of the
		_	through serum	invention (including antibodies and agonists or antagonists of the invention) to regulate serum
			response element	response factors and modulate the expression of genes involved in growth and upregulate the
			in immune cells	function of growth-related genes in many cell types. Exemplary assays for transcription through the

			(such as natural killer cells).	SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992):
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each
				of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used
				according to these assays include the NK-YT cell line, which is a human natural killer cell line with
172	НКАDQ91	797	Production of IL-	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used
			10 and activation	or routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			of T-cells.	agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation
				of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of
				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
				modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed
				and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-
				968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology &
				Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
				reference in their entirety. Exemplary cells that may be used according to these assays include Th2
				cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells
				are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation
				and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and
				asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions
				using peripheral blood lymphocytes isolated from cord blood.
173	HKAEV06	798	Regulation of	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and
			viability and	may be used or routinely modified to assess the ability of polypeptides of the invention (including
			proliferation of	antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of
			pancreatic beta	pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the
			cells.	number of viable cells in culture based on quantitation of the ATP present which signals the
				presence of metabolically active cells. Exemplary assays that may be used or routinely modified to
				test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in:
				Ohtani KI, et al., Endocrinology, 139(1):172-8 (1998); Krautheim A, et al, Exp Clin Endocrinol
				Diabetes, 107 (1):29-34 (1999), the contents of each of which is herein incorporated by reference in

				its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g.,
				through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line
				established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose
				and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs. Lord
				and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
173	HKAEV06	862	Activation of	Assays for the activation of transcription through the AP1 response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through AP1	(including antibodies and agonists or antagonists of the invention) to modulate growth and other cell
			response element	functions. Exemplary assays for transcription through the AP1 response element that may be used
			in immune cells	or routinely modified to test API-response element activity of polypeptides of the invention
			(such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
			-	Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem
				272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al.,
				Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by
				reference in its entirety. Human T cells that may be used according to these assays are publicly
				available (e.g., through the ATCC). Exemplary human T cells that may be used according to these
				assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.
174	HKAFK41	662	Production of	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely
	•		ICAM-1	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or
				routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al.,
				Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365
				(1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163
				(2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that
				may be used according to these assays are publicly available (e.g., through the ATCC) and/or may
				be routinely generated. Exemplary cells that may be used according to these assays include Aortic
				Smooth Muscle Cells (AOSMC); such as bovine AOSMC.
174	HKAFK41	799	Production of IL-6	IL-6 FMAT. IL-6 is produced by T cells and has strong effects on B cells. IL-6 participates in IL-4
				induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6
				induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease,

				plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the
				expression rever is strongly regulated by cytokines, growin factors, and normones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonitis or antagonitis of the invention) to mediate immunomodulation
				and differentiation and modulate T cell proliferation and function. Exemplary assays that test for
				immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation
				and upregulation of a cent prometation and functional activities. Such assays that may be used of routinely modified to test immunomodulatory and diffferentiation activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al.
-				"Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Verhasselt et al., J Immunol
				158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its
			_	entirety. Human dendritic cells that may be used according to these assays may be isolated using
				techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen
				presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate
				and upregulate T cell proliferation and functional activities.
175	HKAFT66	008	Myoblast cell	Assays for muscle cell proliferation are well known in the art and may be used or routinely modified
			proliferation	to assess the ability of polypeptides of the invention (including antibodies and agonists or
			-	antagonists of the invention) to stimulate or inhibit myoblast cell proliferation. Exemplary assays
				for myoblast cell proliferation that may be used or routinely modified to test activity of polypeptides
				and antibodies of the invention (including agonists or antagonists of the invention) include, for
				example, assays disclosed in: Soeta, C., et al. "Possible role for the c-ski gene in the proliferation of
				myogenic cells in regenerating skeletal muscles of rats" Dev Growth Differ Apr;43(2):155-64
				(2001); Ewton DZ, et al., "IGF binding proteins-4, -5 and -6 may play specialized roles during L6
				myoblast proliteration and differentiation. J Endocrinol Mar;144(3):539-53 (1995); and, Pampusch
				MS, et al., Effect of transforming growth factor beta on proliferation of L6 and embryonic porcine
				myogenic cells" J Cell Physiol Jun;143(3):524-8 (1990); the contents of each of which are herein
				incorporated by reference in their entirety. Exemplary myoblast cells that may be used according to
				these assays include the rat myoblast L6 cell line. Rat myoblast L6 cells are an adherent rat
				myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuse to form
				multinucleated myotubes and striated fibers after culture in differentiation media.
175	HKAFT66	800	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or

	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast transcription through GATA-3 in mast cells has been linked to cytokine and chemokine through GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are response element cells (such as mast cells). (such as mast development. Exemplary assays for transcription through the GATA3 response element are president in immune cells). (such as mast development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66.1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 83:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the
	175 HKAFT66

				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
175	HKAFT66	008	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999), Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Tumer et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
176	HKB1E57	801	Regulation of viability and proliferation of pancreatic beta cells.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays

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			include rat LNS-1 cells. LNS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167:
HKFBC53	802	Regulation of transcription of Malic Enzyme in adipocytes	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipoocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays includes the H4IIE rat liver hepatoma cell line.
HKGC027	803	Production of GM-CSF	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage. Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory cytokine. Assays for immunomodulatory proteins that promote the production of GM-CSF are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and modulate the growth and differentiation of leukocytes. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular

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Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach." Chapter 6:138-160 (2000); and Ye et al., J Leukoc Biol (58(2):225-233, the contents of each of which are herein incorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclosed herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor cells and also recognize antibody bound on target cells, via NK Fc receptors, leading to cellmediated cytotoxicity.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the
	Regulation of viability and proliferation of pancreatic beta cells.	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).
	804	808
	HKGDL36	HKISB57
	179	180

			contents of each of which are herein incorporated by reference in its enturety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	
HKISB57	\$08	Regulation of transcription of Malic Enzyme in adipocytes	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipoocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays includes the H4IE rat liver hepatoma cell line.	re : a
HKIYP40	908	Production of IL- 10 and activation	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and	

			of T-cells.	agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL.10, IL.13, IL.5 and IL.6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	s s s
182	HKMLK53	807	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions, they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils." Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils." J Bxp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced	

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				phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.
183	HKMLM11	808	Myoblast cell proliferation	Assays for muscle cell proliferation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit myoblast cell proliferation. Exemplary assays for myoblast cell proliferation that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays disclosed in: Soeta, C., et al. "Possible role for the c-ski gene in the proliferation of myogenic cells in regenerating skeletal muscles of rats" Dev Growth Differ Apr;43(2):155-64 (2001); Ewton DZ, et al., "IGF binding proteins-4, -5 and -6 may play specialized roles during L6 myoblast proliferation and differentiation" J Endocrinol Mar;144(3):539-53 (1995); and, Pampusch MS, et al., "Effect of transforming growth factor beta on proliferation of L6 and embryonic porcine myogenic cells" J Cell Physiol Jun;143(3):524-8 (1990); the contents of each of which are herein incorporated by reference in their entirety. Exemplary myoblast cells that may be used according to these assays include the rat myoblast L6 cell line. Rat myoblast L6 cells are an adherent rat myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuse to form multinucleated myotubes and striated fibers after culture in differentiation media.
184	HKMMW74	808	Regulation of viability and proliferation of pancreatic beta cells.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al.

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				Endocrinology 1992, 130:167.
185	HLDON23	810	Regulation of transcription through the PEPCK promoter in hepatocytes	Assays for the regulation of transcription through the PEPCK promoter are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the PEPCK promoter in a reporter construct and regulate liver gluconeogenesis. Exemplary assays for regulation of transcription through the PEPCK promoter that may be used or routinely modified to test for PEPCK promoter activity (in hepatocytes) of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Lochhead et al., Diabetes 49(6):896-903 (2000); and Yeagley et al., J Biol Chem 275(23):17814-17820 (2000), the contents of each of which is herein incorporated by reference in its entirety. Hepatocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary liver hepatoma cells that may be used according to these assays include H4lle cells, which contain a tyrosine amino transferase that is inducible with glucocorticoids, insulin, or cAMP derivatives.
185	HLDON23	810	Production of VCAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to meaure the upregulation of cell surface VCAM-1 expression in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors, and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.
185	HLDON23	810	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be

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				routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
185	HLDON23	810	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
186	HLDOW79	811	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Brzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the

				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
186	HLDOW79	811	Activation of transcription through NFAT	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			response element in immune cells	response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			(such as mast cells).	invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response
				element that may be used or routinely modified to test NFAT-response element activity of nolvnentides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
	_			Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 63:0342-0340 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-
				7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et
				al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by
	· ·			(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
186	HLDOW79	811	Activation of	Assays for the activation of transcription through the API response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through AP1	(including antibodies and agonists or antagonists of the invention) to modulate growth and other cell
			response element	functions. Exemplary assays for transcription through the API response element that may be used
			in immune cells	or routinely modified to test API-response element activity of polypeptides of the invention
			(such as T-cells).	(including antibodies and agonists of antagonists of the invention) include assays disclosed in Rerger et al., Gene 66:1-10 (1988): Cullen and Malm. Methods in Enzymol 216:362-368 (1992):
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem
				272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al.,
	•			Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by
				reference in its entirety. Human T cells that may be used according to these assays are publicly
				available (e.g., through the ATCC). Exemplary human T cells that may be used according to these
				assays include the SUP1 cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.

186	HLDOW79	811	Activation of transcription	Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through CD28	(including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T
			response element	cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention
			(such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol
				159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Pist China 2010:663 660 (1000), the contents of each of which are herein incommented by reference
		•		BIOI CREM 3(1):332-300 (1998), the contents of each of which are nevel incorporated by reference in its antirety. The letter may be used according to these assays are nublicly available (e.g.
				through the ATCC). Exemplary human T cells that may be used according to these assays include
				the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
186	HLDOW79	811	Activation of	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			transcription	response element are well-known in the art and may be used or routinely modified to assess the
			through NFAT	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			response element	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			in immune cells	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
			(such as T-cells).	element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);
				Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol
				31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J
				Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by
				reference in its entirety. T cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human T cells that may be used according to these assays
				include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
186	HLDOW79	811	Activation of	Assays for the activation of transcription through the NFKB response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through NFKB	(including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription
			response element	factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription
			in immune cells	through the NFKB response element that may be used or rountinely modified to test NFKB-
			(such as T-cells).	response element activity of polypeptides of the invention (including antibodies and agonists or

				antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is, a suspension culture of IL-2 and IL-4 responsive T cells.
187	Н БОР 62	812	Regulation of viability and proliferation of pancreatic beta cells.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
187	HLDQR62	812	Activation of transcription through cAMP response element in immune cells (such as T-cells).	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP and regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117 (1997); and Belkowski et al., I Immunol 161(2):659-665 (1998), the contents of each of which

				are herein incomposated by reference in its entirety. T cells that may be used according to these
				assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used
				according to these assays include the CTLL cell line, which is a suspension culture of IL-2 dependent cytotoxic T cells.
188	нгролл9	813	Regulation of	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and
			viability and	may be used or routinely modified to assess the ability of polypeptides of the invention (including
			proliferation of	antibodies and agonists of antagonists of the invention) to regulate viability and profite around of
			pancreatic pera	number of viable cells in culture based on quantitation of the ATP present which signals the
				presence of metabolically active cells. Exemplary assays that may be used or routinely modified to
				test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in:
				Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology,
				139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the
				contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that
-				may be used according to these assays are publicly available (e.g., through the ATCC) and/or may
				be routinely generated. Exemplary pancreatic cells that may be used according to these assays
				include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated
				from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of
				native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al.
				Endocrinology 1992 130:167.
188	HLDQU79	813	Activation of	Assays for the activation of transcription through the Serum Response Element (SRE) are well-
	,		transcription	known in the art and may be used or routinely modified to assess the ability of polypeptides of the
			through serum	invention (including antibodies and agonists or antagonists of the invention) to regulate the serum
			response element	response factors and modulate the expression of genes involved in growth. Exemplary assays for
			in immune cells	transcription through the SRE that may be used or routinely modified to test SRE activity of the
			(such as T-cells).	polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and
				Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated
				by reference in its entirety. T cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays
				include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic
				activity.

Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.	TNFa FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells, fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and cytotoxic effects on a variety of cells are well known in the art and may be used or routinely
Regulation of viability and proliferation of pancreatic beta cells.	Activation of T-Cell p38 or JNK Signaling Pathway.	Production of TNF alpha by dendritic cells
814	815	816
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189	190	191

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modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, modulate inflammation and cytotoxicity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines such as tumor necrosis factor alpha (TNFa), and the induction or inhibition of an inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Verhasselt et al., Eur J Immunol 28(11):3886-3890 (1198); Dahlen et al., J Immunol 160(7):3585-3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	MIP-1alpha FMAT. Assays for immunomodulatory proteins produced by activated dendritic cells that upregulate monocyte/macrophage and T cell chemotaxis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagofists of the invention) to mediate immunomodulation, modulate chemotaxis, and modulate T cell differentiation. Exemplary assays that test for immunomodulatory proteins evaluate the production of chemokines, such as macrophage inflammatory protein 1 alpha proteins evaluate the production of monocytes/macrophages and T cells. Such assays that may be used or routinely modified to test immunomodulatory and chemotaxis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); Drakes et al., Transp Immunol 8(1):17-29 (2000); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	L6 FMAT. L6 is produced by T cells and has strong effects on B cells. L6 participates in L4
	Production of MIP1alpha	Production of IL-6
	816	816
	HLBD68	HL.BD68
	191	191

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induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6 induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and differentiation and function and function. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	
	Stimulation of insulin secretion from pancreatic beta cells.
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	HLBD68
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				typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
192	н. п. с. с. с. с. с. с. с. с. с. с. с. с. с.	817	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.
192	н.лсQ90	817	Production of TNF alpha by dendritic cells	TINFA FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells, fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and cytotoxic effects on a variety of cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, modulate inflammation and cytotoxicity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines such as tumor necrosis factor alpha (TNFa), and the induction or inhibition of an inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Verhasselt et al., Eur J Immunol 128:13):386-3890 (1198); Dahlen et al., J Immunol 128:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.
192	нгісо90	817	Stimulation of	Assays for measuring calcium flux are well-known in the art and may be used or routinely

modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995); Mogami H, et al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References:
Calcium Flux in pancreatic beta cells.	Stimulation of insulin secretion from pancreatic beta cells.
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				Asfari et al Endocrinology 1992 130:167.
193	нг. фрн. 79	818	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
194	HLTDV50	819	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al, Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
195	HLTE125	820	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays

				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC I cell line which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells
195	HL.TEI25	820	Activation of transcription	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production.
			through NFAT	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			response element	response element are well-known in the art and may be used or routinely modified to assess the
			(such as mast	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
				element that may be used or routinely modified to test NFA1-response element activity of
				polypeptities of the invention (including announces and agonists of annagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998): Cullen and Malm. Methods in
		_		Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De
				Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-
				7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et
				al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
106	תו דעים לל	821	Crimulation of	mast cells. A seave for measuring secretion of insulin are well-known in the art and may be used or routinely
061	TITTI TOO	170	insulin secretion	modified to assess the ability of polyneptides of the invention (including antibodies and agonists or
			from pancreatic	antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is
	•		beta cells.	measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is
				upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component

				in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-
				204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according
				to these assays include fat these feats. These are a solur-addiction of the established from the capacitation cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
197	HLTIP94	822	Stimulation of insulin secretion from pancreatic	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is
			beta cells.	measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component
				in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and
	,			agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al.,
				FEBS Left, 377(2):237-9 (1992); and, Miragila S et. al., Journal of Diomorecular Screening, 4:123-204 (1999), the contents of each of which is herein incorporated by reference in its entirety.
				Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according
				to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics
				typical of native pancreatic beta cells including glucose inducible insulin secretion. References:
		000		Asfari et al. Endocrinology 1992 130:167.
198	HLWAA17	823	Regulation of transcription of	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including
			Malic Enzyme in	antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a
			adipocytes	key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified

				as putative PPAK response elements. ML promoter may also responds to Ar1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipoocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362–368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays includes the H4IIE rat liver hepatoma cell
198	HLWAA17	823	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al., FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
199	HLWAD77	824	Activation of transcription through the EGR (Early Growth Response) element in immune cells (such as B-cells).	Assays for the activation of transcription through the EGR response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate EGR transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the EGR response element that may be used or routinely modified to test EGR response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Richards JD, et al., J Immunol, 166(6):3855-3864 (2001); Dinkel, A, et al., J Exp Med, 188(12):2215-2224 (1998); and, Newton, JS, et al., Eur J Immunol 1996 Apr;26(4):811-816 (1996), the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary epithelial cells that may be used according to these assays include the Raii cell line.
200	HLWA022	825	Production of	MCP-1 FMAT. Assays for immunomodulatory proteins that are produced by a large variety of cells

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and act to induce chemotaxis and activation of monocytes and T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce chemotaxis, and modulate immune cell activation. Exemplary assays that test for immunomodulatory proteins evaluate the production of cell surface markers, such as monocyte chemoattractant protein (MCP), and the activation of monocytes and T cells. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the
MCP-1	Activation of transcription through NFAT response in immune cells (such as T-cells).	Activation of transcription
	825	825
	HLWA022	HLWA022
	200	200

through serum response elemer in immune cells (such as natural killer cells).			
duction of P-1	Production MCP-1	tion	Production MCP-1
ivation of JNK naling Pathway mmune cells ch as incophils).	Activation Signaling in immune (such as		Activation Signaling in immune (such as

201	HLWAY54	826	Upregulation of CD152 and activation of T cells	CD152 FMAT. CD152 (a.k.a. CTLA-4) expression is restricted to activated T cells. CD152 is a negative regulator of T cell proliferation. Reduced CD152 expression has been linked to hyperproliferative and autoimmune diseases. Overexpression of CD152 may lead to impaired immunoresponses. Assays for immunomodulatory proteins important in the maintenance of T cell homeostasis and expressed almost exclusively on CD4+ and CD8+ T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate the activation of T cells, maintain T cell homeostasis, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers, such as CD152, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Curr Opin Immunol 11(3):313-321 (1998),

the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.	Kinase assay. Kinase assays, for example an GSK-3 assays, for PI3 kinase signal transduction that regulate glucose metabolism and cell survival are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit glucose metabolism and cell survival. Exemplary assays for PI3 kinase activity that may be used or routinely modified to test PI3 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Nikoulina et al., Diabetes 49(2):263-271 (2000); and Schreyer et al., Diabetes 48(8):1662-1666 (1999), the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions, they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polyneorides of the invention (including antibodies and aeonists or antagonists
	Activation of Adipocyte PI3 Kinase Signalling Pathway	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).
	827	827
	HLWCF05	HLWCF05
	202	202

				of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-
				activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2:187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR,
				et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosvohorvlation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal
				kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incompared by reference in its entirety.
202	HLWCF05	827	Activation of	Assays for the activation of transcription through the AP1 response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through AP1	(including antibodies and agonists or antagonists of the invention) to modulate growth and other cell
		-	response element	functions. Exemplary assays for transcription through the AP1 response element that may be used
			in immune cells	or routinely modified to test AP1-response element activity of polypeptides of the invention
			(such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem
				272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al.,
				Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by
				reference in its entirety. Human T cells that may be used according to these assays are publicly
				available (e.g., through the ATCC). Exemplary human T cells that may be used according to these
				assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.
202	HLWCF05	827	Activation of	Assays for the activation of transcription through the CD28 response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through CD28	(including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T
			response element	cells. Exemplary assays for transcription through the CD28 response element that may be used or
			in immune cells	routinely modified to test CD28-response element activity of polypeptides of the invention
			(such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol
				159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J
				Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference
				in its entirety. T cells that may be used according to these assays are publicly available (e.g.,

				through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
	HLWCF05	827	Activation of transcription through NFAT response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
202	HLWCF05	827	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-85:6342-6346 (1998); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
203	HLYAC95	828	Production of IFNgamma using a T cells	IFNgamma FMAT. IFNg plays a central role in the immune system and is considered to be a proinflammatory cytokine. IFNg promotes TH1 and inhibits TH2 differentiation; promotes IgG2a and inhibits IgE secretion; induces macrophage activation; and increases MHC expression. Assays for immunomodulatory proteins produced by T cells and NK cells that regulate a variety of

Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res	Nature 410(6824):37-40 (1999); hydrakis JM, prochem Sec Symp Ca. 27-40 (1979), Chang and Main, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to	assess the ability of polypeptides of the invention (including antibodies and agoinsts of antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2:187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR,	et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	Assays for the regulation of transcription through the PEPCK promoter are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the PEPCK promoter in a reporter construct and regulate liver gluconeogenesis. Exemplary assays for regulation of transcription through the PEPCK promoter that may be used or routinely modified to test for	PEPCK promoter activity (in hepatocytes) of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Lochhead et al., Diabetés 49(6):896-903 (2000); and Yeagley et al., J Biol Chem 275(23):17814-17820 (2000), the contents of each of which is herein
Activation of JNK Signaling Pathway in immune cells (such as eosinophils).				Regulation of transcription through the PEPCK promoter in hepatocytes	
829			į	830	
HLYAZ61				HMADK33	
204				205	

				incorporated by reference in its entirety. Hepatocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary liver hepatoma cells that may be used according to these assays include H4lle cells, which contain a tyrosine amino transferase that is inducible with glucocorticoids, insulin, or cAMP derivatives.
206	HMADU73	831	Production of TNF alpha by dendritic cells	TNFa FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells, fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and cytotoxic effects on a variety of cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, modulate inflammation and cytotoxicity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines such as tumor necrosis factor alpha (TNFa), and the induction or inhibition of an inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Verhasselt et al., Eur J Immunol 128(11):3886-3890 (1198); Dahlen et al., J Immunol 160(7):3585-3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.
	HMADU73	831	Production of IL-6	IL-6 FMAT. IL-6 is produced by T cells and has strong effects on B cells. IL-6 participates in IL-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6 induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and differentiation and modulate T cell proliferation and function. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and functional activities. Such assays that may be used or

		·		each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
207	HMAMI15	832	Upregulation of CD152 and activation of T cells	regative regulator of T cell proliferation. Reduced CD152 expression has been linked to hyperproliferative and autoimmune diseases. Overexpression of CD152 may lead to impaired immunoresponses. Assays for immunomodulatory proteins important in the maintenance of T cell homeostasis and expressed almost exclusively on CD4+ and CD8+ T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate the activation of T cells, maintain T cell homeostasis, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers, such as CD152, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Curr Opin Immunol 11(3):294-300 (1999); and Saito T, Curr Opin Immunol 10(3):313-321 (1998), the contents of each of which are herein incoporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cellmediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.
208	HMCFY13	833	Regulation of transcription via DMEF1 response element in	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin

production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed inThai, M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 275(21):16323-8 (2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23566-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362–368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays include the mouse 373-L1 cells are a continuous substrain of 373 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.	Assays for the regulation of transcription through the PEPCK promoter are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the PEPCK promoter in a reporter construct and regulate liver gluconeogenesis. Exemplary assays for regulation of transcription through the PEPCK promoter that may be used or routinely modified to test for PEPCK promoter activity (in hepatocytes) of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Lochhead et al., Diabetes 49(6):896-903 (2000); and Yeagley et al., J Biol Chem 275(23):17814-17820 (2000), the contents of each of which is herein incorporated by reference in its entirety. Hepatocyte cells that may be used according to these assays include H4lle cells, which contain a tyrosine amino transferase that is inducible with glucocorticoids, insulin, or cAMP derivatives.	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
adipocytes and pre-adipocytes	Regulation of transcription through the PEPCK promoter in hepatocytes	Production of ICAM-1
	834	835
·	HMDAB56	HMDAQ29
	209	210

				antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that
	,			may be used according to mese assays are publicly available (e.g., unough use ATCC) among may be routinely generated. Exemplary cells that may be used according to these assays include Aortic Smooth Muscle Cells (AOSMC); such as bovine AOSMC.
210	HMDAQ29	835	Activation of transcription	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through cAMP response element	(including antibodies and agonists or antagonists of the invention) to increase cAMP and regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell
			in immune cells	functions. Exemplary assays for transcription through the cAMP response element that may be used
			(such as T-cells).	or routinely modified to test cAMP-response element activity of polypeptides of the invention
				(including annoones and agoinsts of antagoinsts of the invention) include assays discussed in Berger et al., Gene 66:1-10 (1998): Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-
		_ 		117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which
				are herein incorporated by reference in its entirety. T cells that may be used according to these
				assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used
	•			according to these assays include the CTLL cell line, which is a suspension culture of IL-2
211	HMECK83	836	Activation of	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			transcription	response element are well-known in the art and may be used or routinely modified to assess the
			through NFAT	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			response element	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			in immune cells	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
			(such as T-cells).	element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);
				Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol
				31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J
				Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by

				reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the STDT cell line, which is a suspension culture of H. 2 and H. 4 responsive T cells
212	нмеер18	837	Production of IL6 by primary human aortic smooth muscle or normal human dermal fibroblast cells (without or with costimulation with TNFalpha).	Assay to measure regulation of production of Interleukin-6 (IL-6) by either human aortic smooth muscle cells or normal human dermal fibroblasts minus or plus costimulation with TNFalpha (TNFa). Human aortic smooth muscle cells or normal human dermal fibroblasts may be obtained from commercial sources; these cells are important structural and functional components of blood vessels and connective tissue, respectiviely. Interleukin-6 (IL-6) is a key molecule in chronic inflammation and has been implicated in the progression of atherosclerosis, stroke, arthritis and other vascular and inflammatory diseases. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and production of IL-6.
212	HMEED18	837	Stimulation of Calcium Flux in pancreatic beta cells.	Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) polypeptides of the invention (including antibodies and agonists of al., Biochem J. 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Call Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoids ceptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219:

				547-551: Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343. 1981.
212	HMEED18	837	Upregulation of CD69 and	CD69 FMAT. CD69 is an activation marker that is expressed on activated T cells, B cells, and NK cells. CD69 is not expressed on resting T cells, B cells, or NK cells. CD69 has been found to be
			activation of T	associated with inflammation. Assays for immunomodulatory proteins expressed in T cells, B cells, and leukocytes are well known in the art and may be used or routinely modified to assess the ability
				of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
		_		Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface
				markers, such as CD69, and the activation of T cells. Such assays that may be used or routinely
				and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia
				et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical
				approach" Chapter 6:138-160 (2000); Ferenczi et al., J Autoimmun 14(1):63-78 (200); Werfel et
				(1995); and Afetra et al., Ann Rheum Dis 52(6):457-460 (1993), the contents of each of which are
			-	herein incorporated by reference in its entirety. Human T cells that may be used according to these
				assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T
				cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and
	-			CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be
				preactivated to enhance responsiveness to immunomodulatory factors.
213	HMEFT54	838	Regulation of	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or
			apoptosis in	routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			pancreatic beta	agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis.
			cells.	Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary
				assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity
				of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et
				al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):08/-
				94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:544-7 (2001); Suk K, et al., J Immunol,
				100(7):4461-9 (2001); 1eJeno J, et al., FEBS Lett, 439(2):230-43 (1999); Zhang, 3., et al., FEBS 1 et 485(2-3): 122-126 (2000): Nor et al., J Vasc Res
				37(3); 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the
				contents of each of which are herein incorporated by reference in its entirety. Pancreatic cells that
				may be used according to these assays are publicly available (e.g., through the ATCC) and/or may

				be routinely generated. Exemplary pancreatic cells that may be used according to these assays include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a radiation induced transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1980 77:3519.
214	HMEGF92	839	Production of ICAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Endothelial cells, which are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used in ICAM production assays include human umbilical vein endothelial cells (HUVEC), and are available from commercial sources. The expression of ICAM (CD54),a intergral membrane protein, can be upregulated by cytokines or other factors, and ICAM expression is important in mediating immune and endothelial cell interactions leading to immune and inflammatory responses. Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety.
214	HMEGF92	839	Regulation of apoptosis in pancreatic beta cells.	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):687-94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:S44-7 (2001); Suk K, et al., J Immunol, 166(7):4481-9 (2001); Tejedo J, et al., FEBS Lett, 459(2):238-43 (1999); Zhang, S., et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays

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				include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a radiation induced transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1980 77:3519.
215	HMIAL37	840	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its enturely. Exemplary cells that may be used according to these assays include essinophils. Eosinophils are important in the late stage of allergic reactions, they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-lun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" Clin Exp Immunol; Oct; 122(1):20-7 (2000); Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and vice or easily physphorylation of JUN A-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation of JUN A-terminal kinase and agonist or interest.
215	НМІАГ.37	840	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed

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and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Assays for the regulation (i.e. increases or decreases) of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of eosinophil cells and cell lines. For example, the CellTiter-Gloô Luminescent Cell Viability Assay (Promega Corp., Madison, WI, USA) can be used to measure the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Eosinophils are a type of immune cell important in allergic responses; they are recruited to tissues and mediate the inflammtory response of late stage allergic reaction. Eosinophil cell lines that may be used according to these assays are publicly available and/or may be routinely generated. Exemplary eosinophil cells that may be used according to these assays include EOL-1 Cells.	IFNgamma FMAT. IFNg plays a central role in the immune system and is considered to be a proinflammatory cytokine. IFNg promotes TH1 and inhibits TH2 differentiation; promotes IgG2a and inhibits IgE secretion; induces macrophage activation; and increases MHC expression. Assays for immunomodulatory proteins produced by T cells and NK cells that regulate a variety of inflammatory activities and inhibit TH2 helper cell functions are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, regulate inflammatory activities, modulate TH2 helper cell function, and/or mediate humoral or cellmediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as Interferon gamma (IFNg), and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., I Clin Lab Anal Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal
and 968 The reference are ast usi	Assare of of via via nun nun preeres res res ance assare	H G H S S S S S S S S S S S S S S S S S
	Regulation of viability or proliferation of immune cells (such as human eosinophil EOL-1 cells).	Production of IFNgamma using a T cells
	841	841
	HMKCG09	HMKCG09
	216	216

				8(5):225-233 (1995); Billiau et al., Ann NY Acad Sci 856:22-32 (1998); Boehm et al., Annu Rev Immunol 15:749-795 (1997), and Rheumatology (Oxford) 38(3):214-20 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.
216	HMKCG09	841	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "Thelper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
217	HMMAH60	842	Activation of Natural Killer Cell ERK Signaling Pathway.	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the human natural Exemplary natural killer cells that may be used according to these assays include the human natural

				killer cell lines (for example, NK-YT cells which have cytolytic and cytotoxic activity) or primary NK cells.
217	НММАН60	842	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
218	нморт36	843	Production of IL-13 and activation of T-cells.	Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science;282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness and allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
219	HMSDL37	844	Regulation of viability and	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including

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antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999), Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	IL-6 FMAT. IL-6 is produced by T cells and has strong effects on B cells. IL-6 participates in IL-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6
proliferation of pancreatic beta cells.	Stimulation of insulin secretion from pancreatic beta cells.	Production of IL-6
	845	846
	HMSF126	HMSFS21
	220	221

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247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary rat natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the
-	Activation of transcription through STAT6 response element in immune cells (such as natural killer cells).	Activation of transcription
	847	847
	HMSHS36	HMSHS36
	222	222

·			through NFAT response element in immune cells (such as natural killer cells).	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.
222	HMSHS36	847	Activation of transcription through serum response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.
223	HMSKC04	848	Activation of transcription through AP1 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the AP1 response element are known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the AP1 response element that may be used or routinely modified to test AP1-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al.,

				Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
223	HMSKC04	848	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
223	HMSKC04	848	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of

				polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
223	HMSKC04	848	Activation of transcription through AP1 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the API response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the API response element that may be used or routinely modified to test API-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.
223	HMSKC04	848	Activation of transcription through CD28 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate LL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference

				in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
223	HMSKC04	848	Activation of transcription through GAS response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays
		^		disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 210:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).
223	HMSKC04	848	Activation of transcription through NFAT response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
223	HMSKC04	848	Activation of transcription through STAT6	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or

			response element in immune cells (such as T-cells).	antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
223	HMSKC04	848	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
223	HMSKC04	848	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);

				Aramhuru et al. T Bxn Med 182/3):801-810 (1995): De Boer et al Int J Biochem Cell Biol
				31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J
				Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are multicly available.
				(e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays
				include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic
223	HMSKC04	848	Activation of	Assays for the activation of transcription through the Serum Response Element (SRE) are well-
			transcription	known in the art and may be used or routinely modified to assess the ability of polypeptides of the
			through serum	invention (including antibodies and agonists or antagonists of the invention) to regulate serum
			response element	response factors and modulate the expression of genes involved in growth and upregulate the
			in immune cells	function of growth-related genes in many cell types. Exemplary assays for transcription through the
	-		(such as natural	SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention
			killer cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol
				153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each
				of which are herein incorporated by reference in its entirety. T cells that may be used according to
				these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used
				according to these assays include the NK-YT cell line, which is a human natural killer cell line with
				cytolytic and cytotoxic activity.
224	HMTAD67	849	Production of IL-	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used
			10 and activation	or routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			of T-cells.	agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation
				of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of
				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
			-	modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed
				and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-
				968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology &
				Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
				reference in their entirety. Exemplary cells that may be used according to these assays include Th2
				cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells
				are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation
				and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and

				asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
225	HMVBS81	850	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
226	HMWDC28	851	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics

				typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
227	HMWF165	852	Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed inThai, M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is and, cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is an adherent mouse preadipocyte cell line. Mouse 373-L1 cells are a continuous substrain of 373 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipocy-like conversion under appropriate differentiation culture conditions.
228	HMWFY10	853	Production of IL-8 by immune cells (such as the human EOL-1 eosinophil cells)	Assay that measures the production of the chemokine interleukin-8 (IL-8) from immune cells (such as the EOL-1 human eosinophil cell line) are well known in the art (for example, measurement of IL-8 production by FMAT) and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit. Eosinophils are a type of immune cell important in allergic responses; they are recruited to tissues and mediate the inflammtory response of late stage allergic reaction. IL8 is a strong immunomodulator and may have a potential proinflammatory role in immunological diseases and disorders (such as allergy and asthma).
228	HMWFY10	853	Production of ICAM in endothelial cells	Endothelial cells, which are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used in ICAM production assays include

			(such as human umbilical vein	human umbilical vein endothelial cells (HUVEC), and are available from commercial sources. The expression of ICAM (CD54), a intergral membrane protein, can be upregulated by cytokines or other factors and ICAM expression is important in mediating immine and endothelial cell interactions
				leading to immune and inflammatory responses. Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
				the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1
				expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE. et al., Atherosclerosis, 149(1):99-110 (2000):
				Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J
				Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein inconcersed by reference in its entirety
228	HMWFY10	853	Production of IL-	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used
			10 and activation	or routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			of T-cells.	agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation
				of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of
				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
				modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed
				968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology &
				Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
				reference in their entirety. Exemplary cells that may be used according to these assays include Th2
				cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells
				are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation
				and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and
				asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions
				using peripheral blood lymphocytes isolated from cord blood.
229	HIMWGY65	854	_	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation,
			Cell p38 or JNK	activation, or apoptosis are well known in the art and may be used or routinely modified to assess
			Signaling	the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			Pathway.	invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis.
	- 0.01			Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test
				JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and
				agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem
				379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM,

Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
	Activation of Endothelial Cell p38 or JNK Signaling Pathway.	Insulin Secretion
	854	855
	HMWGY65	HNEEE24
	229	230

		
generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonits or antagonists of the invention) include assays disclosed inThai, M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays include the mouse 373-L1 cell the which is an adherent mouse preadipocyte cell line. Mouse 373-L1 cells are a continuous substrain of 373 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.	
	Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes	Proliferation of immune cells (such as the HMC-1 human mast cell line)
	856	856
	HNFFC43	HNFFC43
	231	231

				throughout the body. Mast cell activation (via immunoglobulin E-antigen, promoted by T helper cell type 2 cytokines) is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Mast cell lines that may be used according to these assays are publicly available and/or may be routinely generated. Exemplary mast cells that may be used according to these assays include HMC-1, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
231	HNFFC43	856		Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
231	HNFFC43	826	Regulation of transcription of Malic Enzyme in adipocytes	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipoocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein

				incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays includes the H4IIE rat liver hepatoma cell line.
232	HNFIU96	857	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention). Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
233	HNFIY77	858	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of

				each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
234	HNFJF07	829	Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed inThai, M.V., et al., J Biol Chem, 273(21):1428-92 (1998); Mora, S., et al., J Biol Chem, 273(21):16323-8 (2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4;275(31):2366-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays include the mouse 3T3-L1 cells are a continuous substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.
234	HNFJF07	859	Regulation of viability and proliferation of pancreatic beta cells.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the

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·			presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al.	9 ti
HNFJF07	859	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.	ic ic
HNFJF07	859	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al.,	or is nt d d

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FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	Assays for the activation of transcription through the AP1 response element are known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the AP1 response element that may be used or routinely modified to test AP1-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell. Cell 89(4):587-596 (1997): and Henderson et al., Mol
Pan AT AT AT AT AT AT AT AT AT AT AT AT AT	A A C C C C C C C C C C C C C C C C C C	The real results of the real real real real real real real rea
•	Activation of transcription through AP1 response element in immune cells (such as T-cells).	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).
	860	098
	HNF1H45	HNFJH45
	235	235

				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
235	HNFJH45	860	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
236	HNGAP93	861	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).

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Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forere et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Bosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils." Clin Exp Immunol; Oct; 122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nirric oxide in eosinophils." Feb 2; 187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep; 104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhance and publi	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52
Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Insulin Secretion
862	863
HNGEO29	HNGFR31
237	238

				(1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HTT115 Cells. HTT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
239	HNGIH43	864	Production of MCP-1	MCP-1 FMAT. Assays for immunomodulatory proteins that are produced by a large variety of cells and act to induce chemotaxis and activation of monocytes and T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce chemotaxis, and modulate immune cell activation. Exemplary assays that test for immunomodulatory proteins evaluate the production of cell surface markers, such as monocyte chemoattractant protein (MCP), and the activation of monocytes and T cells. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.
239	HNGIH43	864	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the

				invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
239	HNGIH43	864	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66.1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1998); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
240	HNGIJ31	865	Activation of transcription through cAMP response element	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP and regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell

functions. Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is a suspension culture of IL-2 dependent cytotoxic T cells.	MCP-1 FMAT. Assays for immunomodulatory proteins that are produced by a large variety of cells and act to induce chemotaxis and activation of monocytes and T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce chemotaxis, and modulate immune cell activation. Exemplary assays that test for immunomodulatory proteins evaluate the production of cell surface markers, such as monocyte chemoattractant protein (MCP), and the activation of cell surface markers, such assays that may be used or routinely modified to test immunomodulatory and diffferentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and
in immune cells (such as T-cells).	Production of MCP-1	Stimulation of insulin secretion from pancreatic beta cells.
	865	865
	HNGIJ31	HNGIJ31
	240	240

agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.		Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase
agonists or antagonists o 277(4 Pt 2):R959-66 (19 FEBS Lett, 377(2):237-5 204 (1999), the contents Pancreatic cells that may ATCC) and/or may be re to these assays include ra cells isolated from an X- typical of native pancrea Asfari et al. Endocrinole	Activation of Kinase assay. Kinase as Skeletal Mucle Cell PI3 Kinase Signalling antibodies and agonists of antibodies and agonists of the cell survival. Exempto to test PI3 kinase-induce agonists or antagonists of 9):1101-1110 (1998); Ni 48(8):1662-1666 (1999), entirety. Rat myoblast controlled that fuses to form muscle, that fuses to form media.	Activation of JNK Kinase assay. JNK kina Signaling Pathway or apoptosis are well kno in immune cells polypeptides of the inverse (such as
	865	998
	HNGIJ31	HNGIQ46
	240	241

	4 6 g i e
may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.	of IL-6 FMAT. IL-6 is produced by T cells and has strong effects on B cells. IL-6 participates in IL-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6 induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and unresulate T cell proliferation and functional activities.
	Production of
	198
	HNGJE50
	242

242	HNGJE50	198	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J., 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1966); and, Miraglia Set. al., Journal of Biomolecular Screening, 4:193-204 (1999); the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATCM-RS1. Refs: Lord and Ashcroft. Biochem. J. 219:
243	HNGJP69	898	Activation of transcription through cAMP response element (CRE) in preadipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Preadipocytes that may be used according to these assays are publicly available (e.g., through the

ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists
	Activation of transcription through serum response element in pre-adipocytes.	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).
	898	868
	HNGJP69	HNGJP69
	243	243

				of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.
243	HNGJP69	898	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
243	HNGJP69	868	Activation of transcription through NFAT response element in immune cells	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the

			(such as mast cells).	invention) to regulate NFAT transcription factors and modulate expression of genes involved in invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Tumer et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
243	HNGJP69	898	Activation of transcription through NFKB response element in immune cells (such as basophils).	This reporter assay measures activation of the NFkB signaling pathway in Ku812 human basophil cell line. Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Marone et al, Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Basophils that may be used according to these assays include Ku812, originally established from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that can be induced to differentiate into mature basophils.
244	HNGKN89	698	Activation of transcription through cAMP response element (CRE) in pre-	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-LI/CRE reporter assay may be used to identify factors that activate

			adipocytes.	the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Preadipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line
				that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
244	HNGKN89	698	Activation of transcription	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
			through GATA-3 response element	production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
		-	in immune cells	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
			cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				invention (including antibodies and agonists or antagonists of the invention) include assays
		_		disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell. Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
				mast cells.

This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	Assays for the regulation of transcription through the PEPCK promoter are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the PEPCK promoter in a reporter construct and regulate liver gluconeogenesis. Exemplary assays for regulation of transcription through the PEPCK promoter that may be used or routinely modified to test for PEPCK promoter activity (in hepatocytes) of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Lochhead et al., Diabetes 49(6):896-903 (2000); and Yeagley et al., J Biol Chem 275(23):17814-17820 (2000), the contents of each of which is herein incorporated by reference in its entirety. Hepatocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary liver hepatoma cells that may be used according to these assays include H4lle cells, which contain a tyrosine amino transferase that is inducible with glucocorticoids, insulin, or cAMP derivatives.
Activation of transcription through NFAT response element in immune cells (such as mast cells).	Regulation of transcription through the PEPCK promoter in hepatocytes
698	870
HNGKN89	HNGND37
244	245

	_ = "0 "
Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995); Mogami H, et al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL-10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL-10, IL-13, IL-5 and IL-6. Factors that induce differentiation and activation of Th2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
Stimulation of Calcium Flux in pancreatic beta cells.	Production of IL- 10 and activation of T-cells.
871	871
HNGOI12	HNGOI12
246	246

247	нинано!	872	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
248	HNHE142	873	Production of GM-CSF	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage. Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory cytokine. Assays for immunomodulatory proteins that promote the production of GM-CSF are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and modulate the growth and differentiation of leukocytes. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Ye et al., J Leukoc Biol (58(2):225-233, the contents of each of which are herein incorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclosed herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor cells and also recognize antibody bound on target cells, via NK Fc receptors, leading to cell-mediated cytotoxicity.
249	HNHEU93	874	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is

		
upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995);Mogami H, et al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Assays for the regulation of transcription through the PEPCK promoter are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention
•	Stimulation of Calcium Flux in pancreatic beta cells.	Regulation of transcription
	875	876
	HNHFM14	HNHNB29
	250	251

			through the PEPCK promoter	(including antibodies and agonists or antagonists of the invention) to activate the PEPCK promoter in a reporter construct and regulate liver gluconeogenesis. Exemplary assays for regulation of transcription through the PEPCK promoter that may be used or routinely modified to lest for	
				PEPCK promoter activity (in hepatocytes) of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998): Cullen and Malm. Methods in Enzymol 216:362-368 (1992): Henthorn et al., Proc Natl	0
	,			Acad Sci USA 85:6342-6346 (1988); Lochhead et al., Diabetes 49(6):896-903 (2000); and Yeagley et al., J Biol Chem 275(23):17814-17820 (2000), the contents of each of which is herein	<u> </u>
				incorporated by reference in its entirety. Hepatocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated.	
				Exemplary liver nepatolina cells that may be used according to mese assays include mane cells, which contain a tyrosine amino transferase that is inducible with glucocorticoids, insulin, or cAMP derivatives.	
	HNHOD46	877	Activation of	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that	
				regulate cell proliferation or differentiation are well known in the art and may be used or routinely	_
			Signaling Pathway	modified to assess the ability of polypeptides of the invention (including antibodies and agonists of antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation.	<u> </u>
				Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK	
				kinase-induced activity of polypeptides of the invention (including antibodies and agonists or subsecutives of the invention) include the assays disclosed in Forrer et al. Biol Chem 379(8-9):1101.	
				1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999);	•
				Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40	
				(2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of	
				which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used	
		_		according to these assays are publicly available (e.g., intough the ATCC). Exemplaty mouse adinocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an	
				adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells	
		_		developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under	
				appropriate differentiation conditions known in the art.	
252	HNHOD46	<i>LL</i> 8	Regulation of	Assays for the regulation of transcription through the DMEF1 response element are well-known in	_
			transcription via	the art and may be used or routinely modified to assess the ability of polypeptides of the invention	_
			DMEF1 response	(including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response	: په
				element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin	lin
			adipocytes and	production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2	~

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			pre-adipocytes	transcription factor and another transcription factor that is required for insulin regulation of Glut4
				expression in skeletal muscle. GLU14 is the primary insulin-responsive glucose transporter in fall and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1
				response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai,
				M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 275(21):16323-8
	•			(2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair
				regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in
				transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988);
				and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is
				herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used
				according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
				generated. Exemplary cells that may be used according to these assays include the mouse 3T3-L1
				cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous
				substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte
-				to adipose-like conversion under appropriate differentiation culture conditions.
252	HNHOD46	877	Activation of	Assays for the activation of transcription through the cAMP response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through cAMP	(including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate
			response element	CREB transcription factors, and modulate expression of genes involved in a wide variety of cell
			(CRE) in pre-	functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate
			adipocytes.	the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in
				differentiation into adipocytes. CRE contains the binding sequence for the transcription factor
		•		CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response
				element that may be used or routinely modified to test cAMP-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
		_		Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);
	. 1			Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923
				(1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-
				adipocytes that may be used according to these assays are publicly available (e.g., through the
				ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used
				according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line
				that is a continuous substrain of 313 fibroblast cells developed through clonal isolation and undergo

				a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the
252	HNHOD46	877	Activation of transcription through serum response element in pre-adipocytes.	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
252	HNHOD46	877	Activation of transcription through cAMP response element in immune cells (such as T-cells).	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP and regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is a suspension culture of IL-2 dependent cytotoxic T cells.
252	HNHOD46	877	Activation of transcription through serum	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum

			response element in immune cells (such as T-cells).	response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.
252	HNHOD46	877	Production of MPlalpha	MIP-lalpha FMAT. Assays for immunomodulatory proteins produced by activated dendritic cells that upregulate monocyte/macrophage and T cell chemotaxis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, modulate chemotaxis, and modulate T cell differentiation. Exemplary assays that test for immunomodulatory proteins evaluate the production of chemokines, such as macrophage inflammatory protein 1 alpha (MIP-1a), and the activation of monocytes/macrophages and T cells. Such assays that may be used or routinely modified to test immunomodulatory and chemotaxis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., J Kumphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); Drakes et al., Transp Immunol 8(1):17-29 (2000); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.
252	HNHOD46	877	Production of IL-6	IL-6 FMAT. IL-6 is produced by T cells and has strong effects on B cells. IL-6 participates in IL-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6 induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in

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the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and differentiation and modulate T cell proliferation and function. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and functional activities. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell
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·	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	Activation
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			transcription through NFAT	line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			response element	response element are well-known in the art and may be used or routinely modified to assess the ability of polyneptides of the invention (including antibodies and agonists or antagonists of the
	, -	_	as m	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De
				boor et al., int J biochem Cell biol 31(10):1221-1230 (1999), All et al., J Illiminiol 103(12):1213-7223 (2000): Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et
				al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
			•	peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
				mast cells.
252	HNHOD46	877	Activation of	Assays for the activation of transcription through the cAMP response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through cAMP	(including antibodies and agonists or antagonists of the invention) to increase cAMP, bind to CREB
			response element	transcription factor, and modulate expression of genes involved in a wide variety of cell functions.
			in immune cells	Exemplary assays for transcription through the cAMP response element that may be used or
			(such as T-cells).	routinely modified to test cAMP-response element activity of polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-
				117 (1997); and Belkowski et al., J Immunol 161(2):659-665 (1998), the contents of each of which
				are herein incorporated by reference in its entirety. T cells that may be used according to these
_				assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used
				according to these assays include the JURKAT cell line, which is a suspension culture of leukemia
				cells that produce IL-2 when stimulated.
252	HNHOD46	877	Activation of	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			transcription	response element are well-known in the art and may be used of fournely modified to assess the

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ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.	This reporter assay measures activation of the NFkB signaling pathway in Ku812 human basophil cell line. Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Marone et al., Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Basophils that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human basophil cell lines that may be used according to these assays include Ku812, originally established from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that can be induced to differentiate into mature basophils.	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide invention. Exemplary assays for transcription through the GAS response element that
response in immune cells (such as T-cells).	Activation of transcription through NFKB response element in immune cells (such as basophils).	Activation of transcription through GAS response element in immine cells
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			(such as T-cells).	may be used or routinely modified to test GAS-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al. Gene 66:1-10 (1998). Cullen and Malm. Methods in Brizymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al.,
				Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the
				cells, such as the MOLT4 cell line, that may be used according to these assays are publicly available
				(e.g., through the ATCC).
252	HNHOD46	877	Activation of	Assays for the activation of transcription through the NFKB response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through NFKB	(including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription
			response element	factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription
			in immune cells	through the NFKB response element that may be used or rountinely modified to test NFKB-
			(such as T-cells).	response element activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen
				and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA
				85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-
				844 (1999), the contents of each of which are herein incorporated by reference in its entirety.
_				Exemplary human T cells, such as the MOLT4, that may be used according to these assays are
				publicly available (e.g., through the ATCC).
252	HNHOD46	877	Activation of	Assays for the activation of transcription through the NFKB response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through NFKB	(including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription
			response element	factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription
			in immune cells	through the NFKB response element that may be used or rountinely modified to test NFKB-
			(such as natural	response element activity of polypeptides of the invention (including antibodies and agonists or
			killer cells).	antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen
				and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA
				85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J
				Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of
				which are herein incorporated by reference in its entirety. NK cells that may be used according to
				these assays are publicly available (e.g., through the ATCC). Exemplary NK cells that may be used
				according to these assays include the NK-YT cell line, which is a human natural killer cell line with
				cytolytic and cytotoxic activity.

252	HNHOD46	877	Activation of transcription through AP1	Assays for the activation of transcription through the API response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell
			response element in immune cells	functions. Exemplary assays for transcription through the API response element that may be used functions. Exemplary assays for transcription through the API response element activity of polypeptides of the invention disclosed in disclosed in
			(Sucil as 1 Colls).	Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem
				272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur I Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by
				reference in its entirety. Human T cells that may be used according to these assays are publicly
				available (e.g., through the AICC). Exemplary numan 1 cells that may be used according to these assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.
252	HNHOD46	877	Activation of	Assays for the activation of transcription through the CD28 response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through CD28	(including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T
	- 3*		response element	cells. Exemplary assays for transcription through the CD28 response element that may be used or
			in immune cells	routinely modified to test CD28-response element activity of polypeptides of the invention
			(sucn as 1-cells).	(including antibodies and agonists of antagonists of the invention) include assays disclosed in Berner of all Gane 66:1-10 (1998): Cullen and Malm Methods in Enzymol 216:362-368 (1992):
				Henthorn et al., Perc Natl Acad Sci 17SA 85:6342-6346 (1988): McGuire and Jacobelli. J Immunol
				159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J
				Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference
				in its entirety. T cells that may be used according to these assays are publicly available (e.g.,
				through the ATCC). Exemplary human T cells that may be used according to these assays include
				the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
252	HINHOD46	811	Activation of	Assays for the activation of transcription through the Gamma Interteron Activation Site (GAS)
			transcription	response element are well-known in the art and may be used or routinely modified to assess the
			through GAS	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			response element	invention) to regulate STAT transcription factors and modulate gene expression involved in a wide
			in immune cells	variety of cell functions. Exemplary assays for transcription through the GAS response element that
			(such as T-cells).	may be used or routinely modified to test GAS-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 60:1-10 (1998); Cullen and Malm, Methods in Enzymol 210:302-

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368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension
	Activation of transcription through NFAT response element in immune cells (such as T-cells).	Activation of transcription through STAT6 response element in immune cells (such as T-cells).
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252	HNHOD46	877	Activation of	Assays for the activation of transcription through the NFKB response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through NFKB	(including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription
			response element	factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription
			in immune cells	through the NFKB response element that may be used or rountinely modified to test NFKB-
			(such as T-cells).	response element activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen
				and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA
				85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-
				844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T
				cells that may be used according to these assays are publicly available (e.g., through the ATCC).
				Exemplary human T cells that may be used according to these assays include the SUPT cell line,
				which is a suspension culture of IL-2 and IL-4 responsive T cells.
252	HNHOD46	218	Activation of	Assays for the activation of transcription through the Serum Response Element (SRE) are well-
			transcription	known in the art and may be used or routinely modified to assess the ability of polypeptides of the
			through serum	invention (including antibodies and agonists or antagonists of the invention) to regulate serum
			response element	response factors and modulate the expression of genes involved in growth and upregulate the
			in immune cells	function of growth-related genes in many cell types. Exemplary assays for transcription through the
			(such as natural	SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention
			killer cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol
				153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each
				of which are herein incorporated by reference in its entirety. T cells that may be used according to
				these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used
				according to these assays include the NK-YT cell line, which is a human natural killer cell line with
				cytolytic and cytotoxic activity.
253	HINHPD10	878	Activation of	Assays for the activation of transcription through the cAMP response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through cAMP	(including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate
			response element	CREB transcription factors, and modulate expression of genes involved in a wide variety of cell
			(CRE) in pre-	functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate
			adipocytes.	the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in

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differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Preadipocytes that may be used according to these assays are publicly available (e.g., through the according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Brzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell
	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	Activation of
	878	878
	HNHPD10	HNHPD10
	253	253

			transcription through NFAT response element in immune cells (such as mast cells).	line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boor et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
254	HNTB126	879	Regulation of apoptosis in pancreatic beta cells.	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):687-94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:S44-7 (2001); Suk K, et al., J Immunol, 166(7):4481-9 (2001); Tejedo J, et al., FEBS Lett, 455(2):238-43 (1999); Zhang, S., et al., FEBS Lett, 455(3):315-20 (1999); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a

				radiation induced transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1980 77:3519.
255	HNTBI57	088	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998), Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
256	HNTBL27	881	Regulation of apoptosis in pancreatic beta cells.	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):687-94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:S44-7 (2001); Suk K, et al., J Immunol, 166(7):4481-9 (2001); Tejedo J, et al., FEBS Lett, 459(2):238-43 (1999); Zhang, S., et al., J Vasc Res Lett, 455(3):315-20 (1999); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the

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				contents of each of which are herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a radiation induced transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1980 77:3519.
256	HNTBL27	881	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
	HNTCE26	882	Production of TNF alpha by dendritic cells	TNFa FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells, fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and cytotoxic effects on a variety of cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, modulate inflammation and cytotoxicity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines such as tumor necrosis factor alpha (TNFa), and the induction or inhibition of an inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Verhasselt et al., Eur J Immunol 28(11):3886-3890 (1198); Dahlen et al., J Immunol 160(7):3585-

				3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.
257	HNTCE26	882	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
257	HNTCE26	887	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
257	HNTCE26	882	Upregulation of CD69 and	CD69 FMAT. CD69 is an activation marker that is expressed on activated T cells, B cells, and NK cells. CD69 is not expressed on resting T cells, B cells, or NK cells. CD69 has been found to be

			activation of T	associated with inflammation. Assays for immunomodulatory proteins expressed in T cells, B cells, and leukocytes are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate the activation of T cells, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers, such as CD69, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ferenczi et al., J Autoimmun 14(1):63-78 (200); Werfel et al., Allergy 52(4):465-469 (1997); Taylor-Fishwick and Siegel, Eur J Immunol 25(12):3215-3221 (1995); and Afetra et al., Ann Rheum Dis 52(6):457-460 (1993), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be	ty, ce to the ce
258	HNTNI01	883	Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adjocytes and pre-adjocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed inThai, M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 270(21):16323-8 (2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) andor may be routinely	a 2 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1

				generated. Exemplary cells that may be used according to these assays include the mouse 3T3-L1 cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous
				substrain of 31.3 indepotatis developed infough cional isolation. These cens undergo a pre-amporyte to adipose-like conversion under appropriate differentiation culture conditions.
258	HNTNI01	883	Activation of transcription	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through cAMP response element	(including antibodies and agonists of antagonists of the invention) to increase calvar, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell
			(CRE) in pre-	functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate
			adipocytes.	the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor
				CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response
				element that may be used or routinely modified to test cAMP-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 83:6342-6346 (1988);
				Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 2/3:917-923
				(1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-
				adipocytes that may be used according to these assays are publicly available (e.g., through the
				ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used
				according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line
				that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo
				a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the
				art.
258	HNTNI01	883	Activation of	Assays for the activation of transcription through the Serum Response Element (SRE) are well-
			transcription	known in the art and may be used or routinely modified to assess the ability of polypeptides of the
-			through serum	invention (including antibodies and agonists or antagonists of the invention) to regulate the serum
			response element	response factors and modulate the expression of genes involved in growth. Exemplary assays for
			in pre-adipocytes.	transcription through the SRE that may be used or routinely modified to test SRE activity of the
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and
				Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated
				by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly

				available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation
				conditions known in the art.
2			transcription through GAS response element in immune cells (such as eosinophils).	response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate gene expression (commonly via STAT transcription factors) involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., I Immunol 155(10):4582-4587 (1995); the contents of each of which are herein incorporated by reference in its entirety. Moreover, exemplary assays that may be used or routinely modified to assess the ability of
				polypeptides of the invention (including antibodies and agonists of antiagonists of the invention) to activate or inhibit activation of immune cells include assays disclosed and/or cited in: Mayumi M., "EoL-1, a human eosinophilic cell line" Leuk Lymphoma; Jun;7(3):243-50 (1992); Bhattacharya S, "Granulocyte macrophage colony-stimulating factor and interleukin-5 activate STAT5 and induce CIS1 mRNA in human peripheral blood eosinophils" Am J Respir Cell Mol Biol; Mar;24(3):312-6 (2001); and, Du J, et al., "Engagement of the CrkL adapter in interleukin-5 signaling in eosinophils" J Biol Chem; Oct 20;275(42):33167-75 (2000); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are a type of immune cell important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammtory response of late stage allergic reaction. Increases in GAS mediated transcription in eosinophils is typically a result of STAT activation, normally a direct consequence of interleukin or other cytokine receptor stimulation (e.g. IL.3, IL.5 or GMCSF).
258	HNTN101	883	Activation of transcription through NFKB response element	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription

			in immune cells (such as EOL1 cells).	through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA
				85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incompared by reference in its entirety. For example, a reporter assay (which
				measures increases in transcription inducible from a NFkB responsive element in EOL-1 cells) may link the NFKB element to a reneorter sene and hinds to the NFKB transcription factor, which is
				upregulated by cytokines and other factors. Exemplary immune cells that may be used according to these assays include eosinophils such as the human BOL-1 cell line of eosinophils. Bosinophils are
				a type of immune cell important in the allergic responses; they are recruited to tissues and mediate the inflammtory response of late stage allergic reaction. Bol-1 is a human eosinophil cell line.
258	HNTNI01	883	Regulation of	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of nolynentides of the invention (including
		_	Malic Enzyme in	antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a
			adipocytes	key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted
				as putative PPAR response elements. ME promoter may also responds to AP1 and other
				transcription factors. Exemplary assays that may be used or routinely modified to test for regulation
				of transcription of Malic Enzyme (in adipoocytes) by polypeptides of the invention (including
				et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol,
				8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et
				al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B.,
				incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are
				publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary
				hepatocytes that may be used according to these assays includes the H4IIE rat liver hepatoma cell
950	UNTNIO	283	Activation of	This renorter assay measures activation of the GATA.3 sionaling pathway in HMC-1 human mast
200	TOTALIANT	200		cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
			through GATA-3	production. Assays for the activation of transcription through the GATA3 response element are
			response element	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of

			in immune cells	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
		_	(such as mast cells).	transcription tactors and modulate expression of mast cell genes important for infinule response development. Exemplary assays for transcription through the GATA3 response element that may be
		-		used or routinely modified to test GATA3-response element activity of polypeptides of the
···				invention (including antibodies and agonists or antagonists of the invention) include assays
· · · · · · · · · · · · · · · · · · ·				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymoi 210:362-
				500 (1992); nellillolli et al., f foc Ivali Acad Sci OSA 63.0342-0340 (1906), f tavell et al., Cold Spring Harb Symp Quant Riol 64-563-571 (1999); Rodriguez-Palmero et al., Eur I Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
		-		peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
+				mast cells.
258 HN	HNTNIOI	883	Activation of	This reporter assay measures activation of the NFAT signaling pathway in FIMC-1 numan mast cell
			transcription	line. Activation of INFA1 in mast cells has been linked to cytokine and chemokine production.
			through NFAT	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			response element	response element are well-known in the art and may be used or routinely modified to assess the
			in immune cells	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
_	•		(such as mast	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
		•	cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
			-	element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De
				Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-
				7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et
				al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by
		-		reference in its entirety. Mast cells that may be used according to these assays are publicly available
_				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
·				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
				mast cells.

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This reporter assay measures activation of the NFkB signaling pathway in HMC-1 human mast cell line. Activation of NFkB in mast cells has been linked to production of certain cytokines, such as LL-6 and LL-9. Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Stassen et al, J Immunol 166(7):4391-8 (2001); and Marquardt and Walker, J Allergy Clin Immunol 105(3):500-5 (2000), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element in immune cells (such as in the human HMC-1 mast cell line) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Sherman, Immunol Rev 179:48-56 (2001); Malaviya and Uckun, I Immunol 168:421-426 (2002); Masuda et al., J Biol Chem 276:26107-26113 (2001), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
Activation of transcription through NFKB response element in immune cells (such as mast cells).	Activation of transcription through STAT6 response element in immune cells (such as mast cells).
883	883
HNTNI01	HNTNI01
258	258

258	HNTNI01	883	Activation of transcription through NFKB response element in immune cells (such as basophils).	This reporter assay measures activation of the NFkB signaling pathway in Ku812 human basophil cell line. Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Marone et al, Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Basophils that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human basophil cell lines that may be used according to these assays include Ku812, originally established from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that
258	HNTNI01	883	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to bind the serum response factor and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9);3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells, such as the ATCC).
258	HNTN101	883	Activation of transcription through STAT6 response element in immune cells	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may

			(such as natural	be used or routinely modified to test STAT6 response element activity of the polypeptides of the
			killer cells).	invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood
				92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur
				J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000),
				the contents of each of which are herein incorporated by reference in its entirety. T cells that may
		-		be used according to these assays are publicly available (e.g., through the ATCC). Exemplary rat
				natural killer cells that may be used according to these assays are publicly available (e.g., through
				the ATCC).
258	HNTNI01	883	Activation of	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS)
			transcription	response element are well-known in the art and may be used or routinely modified to assess the
			through GAS	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
	-		response element	invention) to regulate STAT transcription factors and modulate gene expression involved in a wide
			in immune cells	variety of cell functions. Exemplary assays for transcription through the GAS response element that
			(such as T-cells).	may be used or routinely modified to test GAS-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al.,
				Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the
				contents of each of which are herein incorporated by reference in its entirety. Exemplary human T
				cells, such as the SUPT cell line, that may be used according to these assays are publicly available
				(e.g., through the ATCC).
258	HNTNI01	883	Activation of	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			transcription	response element are well-known in the art and may be used or routinely modified to assess the
			through NFAT	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			response element	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			in immune cells	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
			(such as natural	element that may be used or routinely modified to test NFAT-response element activity of
			killer cells).	polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);
				Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol
				31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J

Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.	Assays for the regulation of transcription through the FAS promoter element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis. FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Methods in Enzymol. 216:362–368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays, such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
	Regulation of transcription through the FAS promoter element in hepatocytes	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).
	884	884
	норргіз	HODDF13
		259

			Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
HODDF13	884	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Tumer et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
HODDN65	882	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 15(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).

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MIP-1alpha FMAT. Assays for immunomodulatory proteins produced by activated dendritic cells that upregulate monocyte/macrophage and T cell chemotaxis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, modulate chemotaxis, and modulate T cell differentiation. Exemplary assays that test for immunomodulatory proteins evaluate the production of chemokines, such as macrophage inflammatory protein 1 alpha (MIP-1a), and the activation of monocytes/macrophages and T cells. Such assays that may be used or routinely modified to test immunomodulatory and chemotaxis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); Drakes et al., Transp Immunol 8(1):17-29 (2000); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and	MCP-1 FMAT. Assays for immunomodulatory proteins that are produced by a large variety of cells and act to induce chemotaxis and activation of monocytes and T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce chemotaxis, and modulate immune cell activation. Exemplary assays that test for immunomodulatory proteins evaluate the production of cell surface markers, such as monocyte chemoattractant protein (MCP), and the activation of monocytes and T cells. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., 'Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R Coll Surg Ednb 45(1):9-19 (2001); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell
Production of MIP1alpha	Production of MCP-1
988	988
HODDN92	НОДДИ НОДДИ НО НОДДИ НО НО НО НО НО НО НО НО НО НО НО НО НО
261	261

				proliferation and functional activities.
261	НОДДИН	988		IL-6 FMAT. IL-6 is produced by T cells and has strong effects on B cells. IL-6 participates in IL-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6 induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and functional activities. Such assays that may be used or routinely modified to test immunomodulatory and differentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., I Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.
261	HODDN92	988	Regulation of transcription through the FAS promoter element in hepatocytes	Assays for the regulation of transcription through the FAS promoter element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis. FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Biochem J, 317 (Pt 1):257-65 (1996); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays,

				such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.
261	HODDN92	886	Activation of transcription	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
			response element	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
			in immune cells (such as mast	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response
			cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
				mast cells.
797	HODDN92	988	Activation of	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell
			transcription	line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production.
			through NFAT	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			response element	response element are well-known in the art and may be used or routinely modified to assess the
			in immune cells	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			(such as mast	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
				element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De
				Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-

				7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
261	НОББИ92	988	Activation of Endothelial Cell p38 or JNK Signaling Pathway.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.
262	HODFN71	887	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T

				cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce L-2 when stimulated.
262	HODEN71	887	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to bind the serum response factor and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells, such as the ATCC).
262	HODFN71	887	Activation of transcription through STAT6 response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary rat natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC).
262	HODFN71	887	Activation of transcription through CD28	Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T

		
cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 83:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-84 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J
response element in immune cells (such as T-cells).	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).
	887	884
	HODFN71	HODFN71
	262	262

Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirery. Mast cells that may be used according to these assays are publicly available
Biol Chem 268(reference in its e (e.g., through the include the NK- activity.	Assays for the activation of tran known in the art and may be use invention (including antibodies response factors and modulate the function of growth-related genes SRE that may be used or routine (including antibodies and agonis Berger et al., Gene 66:1-10 (199 Henthorn et al., Proc Natl Acad 153(9):3862-3873 (1994); and E of which are herein incorporated these assays are publicly availat according to these assays includ cytolytic and cytotoxic activity.	This reporter ass cell line. Actival production. Assa well-known in the invention (in transcription fac development. E used or routinely invention (inclu- disclosed in Ber 368 (1992); Her Spring Harb Sy 29(12):3914-397 Cell Biol 14(6):
	Activation of transcription through serum response element in immune cells (such as natural killer cells).	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).
	887	888
	HODFN71	HOEFV61
	262	263

 <u>·</u>		(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	
889	Regulation of viability and proliferation of pancreatic beta cells.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ohtani KI, et al., Endocrinology, 139(1):172-8 (1998); Krautheim A, et al, Exp Clin Endocrinol Diabetes, 107 (1):29-34 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays are publicly available (e.g., somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	
 688	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to bind the serum response factor and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells, such as the	

MOLT4, that may be used according to these assays are publicly available (e.g., through the ATCC).	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to
	Activation of T-Cell p38 or JNK Signaling Pathway.	Activation of Endothelial Cell p38 or JNK Signaling Pathway.	Activation of JNK Signaling Pathway in immune cells
	068	068	891
	HOFMT75	HOFMT75	HOFNC14
	265	265	266

			1 2 4 2 2 2	and an intitie and manifermetion and anantherin Bramalan occasion for IME binace
			(sucii as	promote of minimited to promote action, activation, and approass. Exemplant assures for strict annual activity that may be used or routinely modified to test INK kinase-induced activity of polypentides
			./	of the invention (including antibodies and agonists or antagonists of the invention) include the
		_		assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res
	-			247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin,
				Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the
				contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that
				may be used according to these assays include eosinophils. Eosinophils are important in the late
				stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of
				late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to
				assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists
				of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in
				eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in
				dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-
				activated protein kinase in human eosinophils." Clin Exp Immunol; Oct;122(1):20-7 (2000);
				Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med;
				Feb 2,187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR,
				et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced
	_			phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal
				kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of
				each of which are herein incorporated by reference in its entirety.
267	НОГОС73	892	Myoblast cell	Assays for muscle cell proliferation are well known in the art and may be used or routinely modified
			proliferation	to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to stimulate or inhibit myoblast cell proliferation. Exemplary assays
				for myoblast cell proliferation that may be used or routinely modified to test activity of polypeptides
				and antibodies of the invention (including agonists or antagonists of the invention) include, for
				example, assays disclosed in: Soeta, C., et al. "Possible role for the c-ski gene in the proliferation of
				myogenic cells in regenerating skeletal muscles of rats" Dev Growth Differ Apr;43(2):155-64
				(2001); Ewton DZ, et al., "IGF binding proteins-4, -5 and -6 may play specialized roles during L6
				myoblast proliferation and differentiation" J Endocrinol Mar;144(3):539-53 (1995); and, Pampusch
				MS, et al., "Effect of transforming growth factor beta on proliferation of L6 and embryonic porcine
				myogenic cells" J Cell Physiol Jun;143(3):524-8 (1990); the contents of each of which are herein
	•			incorporated by reference in their entirety. Exemplary myoblast cells that may be used according to
				these assays include the rat myoblast L6 cell line. Rat myoblast L6 cells are an adherent rat

				myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuse to form multinucleated myotubes and striated fibers after culture in differentiation media.
268	НОССК63	893	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
269	нонву 12	894	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include Aortic Smooth Muscle Cells (AOSMC); such as bovine AOSMC.
270	HONAH29	895	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol

				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
270	HONAH29	895	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1998); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
271	ноовля	896	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et

				al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells
				The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
272	HOSBY40	897	Regulation of transcription through the FAS promoter element	Assays for the regulation of transcription through the FAS promoter element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis.
			in hepatocytes	FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies
				and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Biochem J, 317 (Pt 1):257-65 (1996); Berger, et al., Gene 66:1-10 (1988); and, Cullan B, et al. Methods in Engange 116:362-368 (1999); the contents of each of which is berein
		-		incorporated by reference in its entirety. Hepatocytes that may be used according to these assays, such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.
273	HOSD125	808	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or
				Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that
				may be used according to these assays are publicly available (e.g., through the ATCC) and/or may

				be routinely generated. Exemplary cells that may be used according to these assays include Aortic Smooth Muscle Cells (AOSMC); such as bovine AOSMC.
273	HOSD125	868	Regulation of apoptosis in pancreatic beta cells.	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):687-94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:S44-7 (2001); Suk K, et al., J Immunol, 166(7):4481-9 (2001); Tejedo J, et al., FEBS Lett 485(2:33-43 (1999); Zhang, S., et al., FEBS Lett, 455(3):315-20 (1999); Lee et al., FEBS Lett 485(2:33-43 (1999); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a radiation induced transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1977 74:628;
273	HOSD125	868	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available

(e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an L-2 dependent suspension-culture cell line with cytotoxic activity.	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis IM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays include 373-L1 cells. 373-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 373 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	Kinase assays, for example an Elk-1 kinase assay for ERK signal transduction that regulates cell
	Activation of T-Cell p38 or JNK Signaling Pathway.	Activation of Adipocyte ERK Signaling Pathway	Regulation of
	668	006	900
	HOSFD58	HOUCQ17	ноисо17
	274	275	275

			proliferation and/or differentiation in immune cells (such as mast cells).	proliferation or differentiation, are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Ali H, et al., J Immunol, 165(12):7215-7223 (2000); Tam SY, et al., Blood, 90(5):1807-1820 (1997); Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Berra et al., Biochem Pharmacol 60(8):1171-1178 (2000); Gupta et al., Exp Cell Res 247(2):495-504 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary immune cells that may be used according to these assays include human mast cells such as the HMC-1 cell line.
275	HOUCQ17	006	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998), Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
275	ноисо17	006	Activation of transcription through NFAT	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)

IPBCU51	response element response element are well-known in the art and may be used or routinely modified to assess the in immune cells (such as mast invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	901 Regulation of viability or proliferation of immune cells (such as human eosinophil EOL-1 cells).	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage. Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory cytokine. Assays for immunomodulatory proteins that promote the production of GM-CSF are well
		HPBCU51	HPBCU51 9

				invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and modulate the growth and differentiation of leukocytes. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Ye et al., J Leukoc Biol (58(2):225-233, the contents of each of which are herein incorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclosed herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor cells and also recognize antibody bound on target cells, via NK Fc receptors, leading to cellmediated cytotoxicity.
277	HPDDC77	902	Activation of T-Cell p38 or JNK Signaling Pathway.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
277	HPDDC77	905	Production of IL-2 and activation of T cells	IL-2 FMAT. IL-2 is the principal T cell factor that allows T cell expansion and differentiation into effector cells. Assays for immunomodulatory proteins secreted by TH1 cells that promote T cell and NK cell growth and differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, promote immune cell growth and differentiation, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for

		
immunomodulatory proteins evaluate the production of cytokines, such as IL-2, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Laduda et al., Immunology 94(4):496-502 (1998); and Powell et al., Immunol Rev 165:287-300 (1998), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cellmediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response
	Activation of transcription through NFAT response element in immune cells (such as mast cells).	Regulation of transcription via DMEF1 response
	903	904
	HPEAD48	HPEAD79
		279

			element in	element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMFFI response element is present in the GLUT4 promoter and binds to MFF2.
				transcription factor and another transcription factor that is required for insulin regulation of Glut4
	•		,	expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat
				and muscle tissue. Exemplary assays that may be used of routhely modified to test for DiviEr 1 response element activity (in adipocytes and ore-adipocytes) by polypeptides of the invention
				(including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai,
				M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 275(21):16323-8
				(2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair
				regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in
				and. Cullen. B., et al., Methods in Brzymol. 216:362–368 (1992), the contents of each of which is
				herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used
				according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
				generated. Exemplary cells that may be used according to these assays include the mouse 3T3-L1
				cell line which is an adherent mouse preadipocyte cell line. Mouse 3T3-L1 cells are a continuous
				substrain of 3T3 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte
				to adipose-like conversion under appropriate differentiation culture conditions.
280	HPEBE79	905	Activation of	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast
			transcription	cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine
			through GATA-3	production. Assays for the activation of transcription through the GATA3 response element are
_			response element	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
			in immune cells,	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
			(such as mast	transcription factors and modulate expression of mast cell genes important for immune response
			cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test GATA3-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
	-			disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
	-			Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
			_	reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays

				include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
281	HPIBO15	906	Regulation of viability and proliferation of pancreatic beta cells.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability and proliferation of pancreatic beta cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
281	HPB015	906	Production of IL-6	IL-6 FMAT. IL-6 is produced by T cells and has strong effects on B cells. IL-6 participates in IL-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). IL-6 induces cytotoxic T cells. Deregulated expression of IL-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and differentiation and function and function. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and diffferentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays distincential in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al.,

				each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
283	HPJBK12		Regulation of apoptosis of immune cells (such as mast cells).	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin B -antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001); Yeatman CF 2nd, et al., J Exp Med, 192(8):1093-1103 (2000); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.
283	HPJBK12	806	Activation of Endothelial Cell p38 or JNK Signaling Pathway.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its

				entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.
284	HPMDK28	606	Stimulation of Calcium Flux in pancreatic beta · cells.	Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
285	HPRAL 78	910	Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in fat and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed inThai,

				M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):16323-8 (2000); Liu, M.L., et al., J Biol Chem, 269(45):28514-21 (1994); "Identification of a 30-base pair regulatory element and novel DNA binding protein that regulates the human GLUT4 promoter in transgenic mice", J Biol Chem. 2000 Aug 4;275(31):23666-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Adipocytes and pre-adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include the mouse 373-L1 cell line which is an adherent mouse preadipocyte cell line. Mouse 373-L1 cells are a continuous substrain of 373 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte
286	HPRBC80	911	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
286	HPRBC80	911	Activation of transcription through NFAT response element	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the

			in immune cells (such as mast	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in	
			cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response	
				element that may be used or routinely modified to test NFAT-response element activity of	
				polypeptides of the invention (including anticodies and agoinsts of antagoinsts of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998): Cullen and Malm. Methods in	
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De	
		•		Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-	
				7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et	ಕ
				al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by	
				reference in its entirety. Mast cells that may be used according to these assays are publicly available	ble
-				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays	s/
				include the HMC-1 cell line, which is an immature human mast cell line established from the	
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature	ire
				mast cells.	
286	HPRBC80	911	Activation of	Assays for the activation of transcription through the AP1 response element are well-known in the	<u> </u>
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention	
_			through AP1	(including antibodies and agonists or antagonists of the invention) to modulate growth and other cell	<u>ا</u>
			response element	functions. Exemplary assays for transcription through the AP1 response element that may be used	-
			in immune cells	or routinely modified to test AP1-response element activity of polypeptides of the invention	
			(such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in	
				Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);	
			•	Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem	
				272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al.,	al.,
				Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by	
				reference in its entirety. Human T cells that may be used according to these assays are publicly	
				available (e.g., through the ATCC). Exemplary human T cells that may be used according to these	မွ
				assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.	ne.
286	HPRBC80	911	Activation of	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)	
			transcription	response element are well-known in the art and may be used or routinely modified to assess the	
-			through NFAT	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the	
			response element	invention) to regulate NFAT transcription factors and modulate expression of genes involved in	
			in immune cells	immunomodulatory functions. Exemplary assays for transcription through the NFAT response	
			(such as T-cells).	element that may be used or routinely modified to test NFAT-response element activity of	

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polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Hand Chem 268(190):4285-14393 (1993); the Contents of each of which are herein incompared by
	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).
·	911	911
	HPRBC80	HPRBC80
	286	286

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reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma "Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast
	Activation of transcription through serum response element in immune cells (such as natural killer cells).	Production of IL- 10 and activation of T-cells.	Activation of
	911	912	913
	HPRBC80	HPRSB76	HPTVX32
	286	287	288

			transcription	rell line Activation of CATA 3 in mast cells has been linked to cotokine and chemokine
			through GATA-3	production. Assays for the activation of transcription through the GATA3 response element are
			response element	well-known in the art and may be used or routinely modified to assess the ability of polypeptides of
			in immune cells	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3
	•		(such as mast	transcription factors and modulate expression of mast cell genes important for immune response
•	-		cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test GATA3-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
		_		29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
		_		include the HMC-1 cell line, which is an immature human mast cell line established from the
				accipant blood of a nationst with most call lankamia and ashibite many characteristics of immature
				peripheral blood of a patient with mast cell leukenina, and exindits many characteristics of miniature
280	HPV A ROA	014	Activation of	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
707	LOCAL TIT		trent varion of	Assures the meaning that the contract of the c
			transcription	response element are well-known in the art and may be used of fourther to assess the
			through NFAT	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
_			response element	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			in immune cells	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
			(such as T-cells).	element that may be used or routinely modified to test NFAT-response element activity of
		_		polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);
				Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol
				31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J
				Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by
				reference in its entirety. T cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human T cells that may be used according to these assays
				include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
290	HPWAY46	915	Activation of	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast

			transcription through GATA-3	cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are
		_ _	response element in immune cells	Well-known in the art and may be used of fourney modified to assess the ability of polypephides of the invention (including antibodies and agonists of antagonists of the invention) to regulate GATA3
			(such as mast	transcription factors and modulate expression of mast cell genes important for immune response
			cens).	used or routinely modified to test GATA3-response element activity of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
				mast cells,
290	HPWAY46	915	Activation of	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell
			transcription	line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production.
			through NFAT	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			response element	response element are well-known in the art and may be used or routinely modified to assess the
			in immune cells	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			(such as mast	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
				element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De
				Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-
				7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et
				al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays

				include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
290	HPWAY46	915	Activation of transcription through CD28 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the CD28 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T cells. Exemplary assays for transcription through the CD28 response element that may be used or routinely modified to test CD28-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol 159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.
290	HPWAY46	915	Activation of transcription through GAS response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).
290	HPWAY46	915	Activation of transcription through STAT6 response element	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of

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Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.	Kinase assay. INK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of neuronal genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Gill 1S, et al., Neurobiol Dis, 7(4):448-461 (2000); Tamatani M, et al., J Biol Chem, 274(13):8531-8538 (1999); Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al., Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Neuronal cells that may be used
	Activation of T-Cell p38 or JNK Signaling Pathway.	Activation of transcription through NFKB response element in neuronal cells (such as SKNMC cells).
	918	918
	HPZAB47	HPZAB47
	293	293

according to these assays are publicly available (e.g., through the ATCC). Exemplary neuronal cells that may be used according to these assays include the SKNMC neuronal cell line.	CD152 FMAT. CD152 (a.k.a. CTLA.4) expression is restricted to activated T cells. CD152 is a negative regulator of T cell proliferation. Reduced CD152 expression has been linked to hyperproliferative and autoimmune diseases. Overexpression of CD152 may lead to impaired immunoresponses. Assays for immunomodulatory proteins important in the maintenance of T cell homeostasis and expressed almost exclusively on CD4+ and CD8+ T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention of T cells, maintain T cell homeostasis, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers, such as CD152, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Curr Opin Immunol 11(3):294-300 (1999); and Saito T, Curr Opin Immunol 10(3):313-321 (1998), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are
	Upregulation of CD152 and activation of T cells	Activation of T-Cell p38 or JNK Signaling Pathway.
	918	616
•	HPZAB47	HRAAB15
	293	294

				publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
294	HRAAB15	616	Production of IFNgamma using a T cells	IFNgamma FMAT. IFNg plays a central role in the immune system and is considered to be a proinflammatory cytokine. IFNg promotes TH1 and inhibits TH2 differentiation; promotes IgG2a and inhibits IgE secretion; induces macrophage activation; and increases MHC expression. Assays for immunomodulatory proteins produced by T cells and NK cells that regulate a variety of inflammatory activities and inhibit TH2 helper cell functions are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, regulate inflammatory activities, modulate TH2 helper cell function, and/or mediate humoral or cellmediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as Interferon gamma (IFNg), and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal 8(5):225-233 (1995); Billiau et al., Ann NY Acad Sci 856:22-32 (1998); Boehm et al., Annu Rev Immunol 15:749-795 (1997), and Rheumatology (Oxford) 38(3):214-20 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T
				Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.
295	HRABA80	920	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52

				(1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219:
295	HRABA80	920	Activation of Endothelial Cell ERK Signaling Pathway.	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Berra et al., Biochem Pharmacol 60(8):1171-1178 (2000); Gupta et al., Exp Cell Res 247(2):495-504 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are endothelial cells which line venous blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.
295	HRABA80	920	Upregulation of CD152 and activation of T cells	CD152 FMAT. CD152 (a.k.a. CTLA-4) expression is restricted to activated T cells. CD152 is a negative regulator of T cell proliferation. Reduced CD152 expression has been linked to hyperproliferative and autoimmune diseases. Overexpression of CD152 may lead to impaired immunoresponses. Assays for immunomodulatory proteins important in the maintenance of T cell homeostasis and expressed almost exclusively on CD4+ and CD8+ T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate the activation of T cells, maintain T cell homeostasis, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers, such as CD152, and the activation of T cells. Such assays that may be used or routinely modified to

				Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
296	HRACD15	921	Regulation of apoptosis of immune cells (such as mast cells).	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate caspase protease-mediated apoptosis in immune cells (such as, for example, in mast cells). Mast cells are found in connective and mucosal tissues throughout the body, and their activation via immunoglobulin E-antigen, promoted by T helper cell type 2 cytokines, is an important component of allergic disease. Dysregulation of mast cell apoptosis may play a role in allergic disease and mast cell tumor survival. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity induced by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Masuda A, et al., J Biol Chem, 276(28):26107-26113 (2001); Veatman CF 2nd, et al., J Exp Med, 192(8):1093-1103 (2000); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays are publicly available (e.g., through commercial sources). Exemplary immune cells that may be used according to these assays include mast cells such as the HMC human mast cell line.
297	HRACJ35	922	Regulation of transcription of Malic Enzyme in hepatocytes	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in hepatocytes) by polypeptides of the invention (including

				antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays includes the mouse 3T3-L1 cell line. 3T3-L1 is a mouse preadipocyte cell line (adherent). It is a continuous substrain of 3T3 fibroblasts developed through clonal isolation. Cells undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation culture conditions.
297	HRACJ35	922	Production of VCAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to meaure the upregulation of cell surface VCAM-1 expression in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors, and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.
298	HRGBL78	923	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the

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ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995);Mogami H, et al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):687-94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:S44-7 (2001); Suk K, et al., J Immunol, 166(7):4481-9 (2001); Tejedo J, et al., FEBS Lett, 459(2):238-43 (1999); Zhang, S., et al., FEBS
	Stimulation of Calcium Flux in pancreatic beta cells.	Regulation of apoptosis in pancreatic beta cells.
	924	925
	HROAJ39	HROBD68
	299	300

Lett, 455(3):315-20 (1999); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a radiation induced transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1980 77:3519.		Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biocham Soc Suma 64-20 48 (1900); Chang and Karin Nature 410(6824):3740 (2001); and Coha
Lett, 455(3) 37(3): 209-; contents of may be use be routinely include RID radiation in hormones, a	Production of IL- 10 and activation of T-cells. of T-cells. polypeptide modulate II and/or cited 968 (2000), Therapeutic reference in cells. IL10 are a class c and activati	Activation of T- Cell p38 or JNK Signaling Pathway. Exemplary JNK and p5 agonists or 379(8-9):11
	926	927
	HSAVD46	нзаун65
	301	302

				incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
303	HSAWD74	928	Regulation of transcription via DMEF1 response element in adipocytes and pre-adipocytes	Assays for the regulation of transcription through the DMEF1 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the DMEF1 response element in a reporter construct (such as that containing the GLUT4 promoter) and to regulate insulin production. The DMEF1 response element is present in the GLUT4 promoter and binds to MEF2 transcription factor and another transcription factor that is required for insulin regulation of Glut4 expression in skeletal muscle. GLUT4 is the primary insulin-responsive glucose transporter in far and muscle tissue. Exemplary assays that may be used or routinely modified to test for DMEF1 response element activity (in adipocytes and pre-adipocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Thai, M.V., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):14285-92 (1998); Mora, S., et al., J Biol Chem, 273(23):123666-73; Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is an adherent mouse preadipocyte cell line. Mouse 373-L1 cells are a continuous substrain of 373 fibroblasts developed through clonal isolation. These cells undergo a pre-adipocyte to adipocyte conversion under appropriate differentiation culture conditions.
303	HSAWD74	928	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)

				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
304	HSAWZ41	929	Activation of transcription through cAMP response element (CRE) in preadipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-LI/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Preadipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 373-L1 cells. 373-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 373 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
304	HSAWZ41	929	Activation of transcription through AP1 response element	Assays for the activation of transcription through the AP1 response element are known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate growth and other cell functions. Exemplary assays for transcription through the AP1 response element that may be used or routinely

			in immune cells (such as T-cells).	modified to test AP1-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem 272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al., Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
304	HSA WZ41	929	Activation of transcription through NFKB response element in immune cells (such as EOL1 cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al., Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. For example, a reporter assay (which measures increases in transcription inducible from a NFKB responsive element in EOL-1 cells) may link the NFKB element to a repeorter gene and binds to the NFKB transcription factor, which is upregulated by cytokines and other factors. Exemplary immune cells that may be used according to these assays include eosinophils such as the human EOL-1 cell line of eosinophils. Eosinophils are a type of immune cell important in the allergic responses; they are recruited to tissues and mediate the inflammtory response of late stage allergic reaction. Eol-1 is a human eosinophil cell line.
304	HSAWZ41	929	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the

				invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
304	HSAWZ41	929	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cells in a second carried to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
304	HSAWZ41	929	Activation of transcription through GAS response element	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide

			in immune cells (such as T-cells).	variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and aconists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al.,
	•			blood 93(0):1300-1931 (1939); and remunen et al., Junimulot 153(10):4502-4507 (1935), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T
				cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).
304	HSAWZ41	929	Activation of	Assays for the activation of transcription through the Signal Transducers and Activators of
			transcription	Transcription (STAT6) response element are well-known in the art and may be used or routinely
			through STAT6	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
			response element	antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of
			in immune cells	multiple genes. Exemplary assays for transcription through the STA16 response element that may
			(such as T-cells).	be used or routinely modified to test STA16 response element activity of the polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood
				92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur
				J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000),
				the contents of each of which are herein incorporated by reference in its entirety. T cells that may
				be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T
				cells that may be used according to these assays include the SUPT cell line, which is a suspension
				culture of IL-2 and IL-4 responsive T cells.
304	HSAWZ41	929	Activation of	Assays for the activation of transcription through the Serum Response Element (SRE) are well-
			transcription	known in the art and may be used or routinely modified to assess the ability of polypeptides of the
			through serum	invention (including antibodies and agonists or antagonists of the invention) to regulate serum
	•		response element	response factors and modulate the expression of genes involved in growth and upregulate the
			in immune cells	function of growth-related genes in many cell types. Exemplary assays for transcription through the
		_	(such as natural	SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention
			killer cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol
				153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each

				of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.
305	HSDAJ46	930	Activation of transcription through NFAT response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in innumunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aramburu et al., J Exp Med 182(3):801-810 (1995); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human natural killer cell line with cytolytic and cytotoxic activity.
306	HSDEK49	931	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 83:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.
306	HSDEK49	931	Regulation of	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be

			transcription of Malic Enzyme in adipocytes	used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other
		·· · · · · · · · · · · · · · · · · · ·		transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipoocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol,
				8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein
		- ···		incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays includes the H4IIE rat liver hepatoma cell line.
307	HSDEZ20	932	Activation of JNK Signaling Pathway	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of
			(such as	polypeptides of the invention (including antibodies and agonists of antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase
			eosinophils).	of the invention (including antibodies and agonists or antagonists of the invention) include the
	- 10			247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, No. 1000000000000000000000000000000000000
				nature 410(0624):37-40 (2001); and Copb Mit, frog Biopnys Mot Biot 71(3-4):475-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that
				may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of
				late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to
				assess the ability of polypeptides of the invention (including antibodies and agomsts of antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in
				eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in
				dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen- activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000);

				Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR,
A12-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-				et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of each of which are herein incorporated by reference in its entirety.
308	HSDF126	933	Regulation of transcription through the PEPCK promoter	Assays for the regulation of transcription through the PEPCK promoter are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the PEPCK promoter in a reporter construct and regulate liver gluconeogenesis. Exemplary assays for regulation of
			in hepatocytes	transcription through the PEPCK promoter that may be used or routinely modified to test for PEPCK promoter activity (in hepatocytes) of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1908). Cultar and Malm Markeds in Engannal 216:353-358 (1902). Heathorn at al., Droc Notl
		-		Acad Sci USA 85:6342-6346 (1988); Lochhead et al., Diabetes 49(6):896-903 (2000); and Yeagley et al., J Biol Chem 275(23):17814-17820 (2000), the contents of each of which is herein
				incorporated by reference in its entirety. Hepatocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary liver hepatoma cells that may be used according to these assays include H4lle cells.
				which contain a tyrosine amino transferase that is inducible with glucocorticoids, insulin, or cAMP derivatives.
309	HSDFW45	934	Activation of transcription	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through cAMP response element	(including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell
<u> </u>			(CRE) in pre- adipocytes.	functions. For example, a 313-L1/CKE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adjacents.
				CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response
				element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 210:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 83:0342-0346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923

				dipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be used according to these assays are publicly available (e.g., through the according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
309	HSDFW45	934	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
309	HSDFW45	934	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of

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	·			polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
310	HSDJA15	935		Kinase assay. Kinase assays, for example an GSK-3 assays, for P13 kinase signal transduction that regulate glucose metabolism and cell survival are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit glucose metabolism and cell survival. Exemplary assays for P13 kinase activity that may be used or routinely modified to test P13 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Nikoulina et al., Diabetes 49(2):263-271 (2000); and Schreyer et al., Diabetes 48(8):1662-1666 (1999), the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
310	HSDJA15	935	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays

				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
310	HSDJA15	935	Production of IL-5	IL-5 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells, mast cells, basophils, and eosinophils that stimulate eosinophil function and B cell Ig production and promote polarization of CD4+ cells into TH2 cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation, stimulate immune cell function, modulate B cell Ig production, modulate immune cell polarization, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-5, and the stimulation of eosinophil function and B cell Ig production. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Ohshima et al., J Allergy Clin Immunol 106(1 Pt 2):558-564 (2000); and Koning et al., Cytokine 9(6):427-436 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomulatory factors.
311	HSDJL42	936	Activation of transcription through NFAT response element in immune cells	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the

				to adinose-like conversion under appropriate differentiation culture conditions
				to authose-line conversion under appropriate universitation current conditions.
312	HSDSB09	937	Activation of	Assays for the activation of transcription through the cAMP response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through cAMP	(including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate
			response element	CREB transcription factors, and modulate expression of genes involved in a wide variety of cell
			(CRE) in pre-	functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate
			adipocytes.	the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in
				differentiation into adipocytes. CRE contains the binding sequence for the transcription factor
	·			CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response
				element that may be used or routinely modified to test cAMP-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988);
				Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923
				(1998), the contents of each of which are herein incorporated by reference in its entirety. Pre-
				adipocytes that may be used according to these assays are publicly available (e.g., through the
				ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used
_				according to these assays include 3T3-L1 cells, 3T3-L1 is an adherent mouse preadipocyte cell line
				that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo
	-			a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the
				art.
312	HSDSB09	937	Activation of	Assays for the activation of transcription through the Serum Response Element (SRE) are well-
	-		transcription	known in the art and may be used or routinely modified to assess the ability of polypeptides of the
			through serum	invention (including antibodies and agonists or antagonists of the invention) to regulate the serum
			response element	response factors and modulate the expression of genes involved in growth. Exemplary assays for
			in pre-adipocytes.	transcription through the SRE that may be used or routinely modified to test SRE activity of the
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and
				Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated
				by reference in its entirety. Pre-adipocytes that may be used according to these assays are publicly
				available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte
				cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse
				preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal

				isologic and underses a new adjustants to adjust a like construction and an analysis of the animities
				conditions known in the art.
312	HSDSB09	937	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998), Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.
312	HSDSB09	937	Regulation of transcription of Malic Enzyme in adipocytes	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipocoytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays includes the H4IIE rat liver hepatoma cell line.
312	HSDSB09	937	Stimulation of Calcium Flux in	Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or

			pancreatic beta cells.	antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995);Mogami H, et al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
312	HSDSB09	937	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the

				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
312	HSDSB09	937	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention). Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
312	HSDSB09	937	Activation of transcription through NFKB response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFkB signaling pathway in HMC-1 human mast cell line. Activation of NFkB in mast cells has been linked to production of certain cytokines, such as IL-6 and IL-9. Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Stassen et al, J Immunol 166(7):4391-8 (2001); and Marquardt and Walker, J Allergy Clin Immunol 105(3):500-5 (2000), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are

	10 10 10 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	or nt nt dd dd 3-,
publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element in immune cells (such as in the human HMC-1 mast cell line) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Sherman, Immunol Rev 179:48-56 (2001); Malaviya and Uckun, J Immunol 168:421-426 (2002); Masuda et al., J Biol Chem 276:26107-26113 (2001), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety.
publicly accordin establish characte	Assays I Inc) are polypep polypep regulate assays f to test S and ago (1998); Acad Sc Uckun, and Mas incorpoi publicly accordire establish	Assays I modified antagon measure upregula in diabe insulin sagonists 277(4 P P FEBS L 204 (199 Pancrea
	Activation of transcription through STAT6 response element in immune cells (such as mast cells).	Stimulation of insulin secretion from pancreatic beta cells.
	937	937
	HSDSB09	HSDSB09
	312	312

				to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
312	HSDSB09	937	Activation of transcription through NFKB response element in immune cells (such as basophils).	This reporter assay measures activation of the NFkB signaling pathway in Ku812 human basophil cell line. Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Marone et al, Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Basophils that may be used according to these assays include Ku812, originally established from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that can be induced to differentiate into mature basophils.
312	HSDSB09	937	Activation of transcription through STAT6 response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.

710			transcription through serum response element in immune cells (such as natural killer cells).	known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with
313	HSDSE75	938	Myoblast cell proliferation	Assays for muscle cell proliferation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit myoblast cell proliferation. Exemplary assays for myoblast cell proliferation that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays disclosed in: Soeta, C., et al. "Possible role for the c-ski gene in the proliferation of myogenic cells in regenerating skeletal muscles of rats" Dev Growth Differ Apr;43(2):155-64 (2001); Ewton DZ, et al., "IGF binding proteins-4, -5 and -6 may play specialized roles during L6 myoblast proliferation and differentiation" J Endocrinol Mar;144(3):539-53 (1995); and, Pampusch MS, et al., "Effect of transforming growth factor beta on proliferation of L6 and embryonic porcine myogenic cells." J Cell Physiol Jun; 143(3):524-8 (1990); the contents of each of which are herein incorporated by reference in their entirety. Exemplary myoblast cells that may be used according to these assays include the rat myoblast L6 cell line. Rat myoblast L6 cells are an adherent rat myoblast cell line, isolated from primary cultures of rat thigh muscle, that fuse to form multinucleated myotubes and striated fibers after culture in differentiation media.
313	HSDSE75	938	Production of IL-6	II-6 FMAT. II-6 is produced by T cells and has strong effects on B cells. II-6 participates in II-4 induced IgE production and increases IgA production (IgA plays a role in mucosal immunity). II-6 induces cytotoxic T cells. Deregulated expression of II-6 has been linked to autoimmune disease, plasmacytomas, myelomas, and chronic hyperproliferative diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where the

				expression level is strongly regulated by cytokines, growth factors, and hormones are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and differentiation and modulate T cell proliferation and function. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as IL-6, and the stimulation and upregulation of T cell proliferation and functional activities. Such assays that may be used or routinely modified to test immunomodulatory and diffferentiation activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell proliferation and functional activities.
314	HSFAM31	939	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics, 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL 10, IL 13, IL.5 and IL.6. Factors that induce differentiation and activation of Th2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
315	HSICV24	940	Activation of transcription through NFAT response element	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the

			in immune cells (such as mast cells).	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66.1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-
				7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al. 1 Fron Med 188:577-537 (1998) the contents of each of which are herein incomposated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
.316	HSIDJ81	941	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely
				antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is
				measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is
				upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component
				in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of isolity eacration from papersatic calls) by polynomides of the invention (including antibodies and
				agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J,
				47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et
				al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52
				(1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of
				according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
				generated. Exemplary pancreatic cells that may be used according to these assays include HITT15
				Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells
				transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors.
				The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by
				somatostatin or glucocorticoids. ATTC# CRL-1777 Refs. Lord and Ashcroft. Biochem. J. 219:
				547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.

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Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of neuronal genes. Exemplary assays for transcription through the NFKB response element that may be used or routinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Gill JS, et al., Neurobiol Dis, 7(4):448-461 (2000); Tamatani M, et al., J Biol Chem, 274(13):8531-8538 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al., Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Neuronal cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary neuronal cells that may be used according to these assays include the SKNMC neuronal cell line.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Bur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell
Activation of transcription through NFKB response element in neuronal cells (such as SKNMC cells).	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	Activation of
941	942	942
HSIDJ81	HSKCP69	HSKCP69
316	317	317

			transcription through NFAT	line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			respónse element in immune cells	response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			(such as mast	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
				element that may be used of fourmery modulied to test INFA 1-195poinse element activity of polymentides of the invention (including antibodies and agonists of antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De
				Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-
				7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et
				al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
				mast cells.
318	HSKDA27	943	Production of	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and
			GM-CSF	fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage
				progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage.
				Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and
				monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory
				cytokine. Assays for immunomodulatory proteins that promote the production of GM-CSF are well
				known in the art and may be used or routinely modified to assess the ability of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) to mediate
				immunomodulation and modulate the growth and differentiation of leukocytes. Exemplary assays
				that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF,
				and the activation of T cells. Such assays that may be used or routinely modified to test
				immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular
				Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-
				160 (2000); and Ye et al., J Leukoc Biol (58(2):225-233, the contents of each of which are herein
				incorporated by reference in its entirety. Natural killer cells that may be used according to these

				assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclosed herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor cells and also recognize antibody bound on target cells, via NK Fc receptors, leading to cellmediated cytotoxicity.
318	HSKDA27	943	Regulation of apoptosis in pancreatic beta cells.	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):687-94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:S44-7 (2001); Suk K, et al., J Immunol, 166(7):4481-9 (2001); Tejedo J, et al., FEBS Lett, 455(2):238-43 (1999); Zhang, S., et al., FEBS Lett, 455(3):315-20 (1999); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a radiation induced transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1977 74:628;
319	HSKGN81	944	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-

				204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
320	HSKHZ81	945	Activation of transcription through cAMP response element (CRE) in preadipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-LI/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Preadipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
320	HSKHZ81	945	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be

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routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage. Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory cytokine. Assays for immunomodulatory proteins that promote the production of GM-CSF are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and modulatory proteins evaluate the production of cytokines, such as GM-CSF, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Ye et al., J Leukoc Biol (58(2):225-233, the contents of each of which are herein incorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclosed herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor cells and also recognize antibody bound on target cells, via NK Fc receptors, leading to cell-mediated cytotoxic ty.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety.
	Production of GM-CSF	Stimulation of insulin secretion from pancreatic beta cells.
	946	947
,	HSLJG37	HSNAD72
	321	322

				and/or may be routinely generated. Exemplary eosinophil cells that may be used according to these assays include EOL-1 Cells.
324	HSQCM10	949	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL 10, IL 13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
325	HSQEO84	950	Regulation of viability or proliferation of immune cells (such as human eosinophil EOL-1 cells).	Assays for the regulation (i.e. increases or decreases) of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of eosinophil cells and cell lines. For example, the CellTiter-Gloô Luminescent Cell Viability Assay (Promega Corp., Madison, WI, USA) can be used to measure the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Eosinophils are a type of immune cell important in allergic responses; they are recruited to tissues and mediate the inflammtory response of late stage allergic reaction. Eosinophil cell lines that may be used according to these assays are publicly available and/or may be routinely generated. Exemplary eosinophil cells that may be used according to these assays include EOL-1 Cells.
325	нѕQЕО84	950	Activation of transcription through GATA-3 response element in immune cells (such as mast	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response

			cells).	development. Exemplary assays for transcription through the GATA3 response element that may be
				used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol
				29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by
		_		reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
325	HSQEO84	950	Activation of	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell
			transcription	line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production.
			through NFAT	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			response element	response element are well-known in the art and may be used or routinely modified to assess the
-			in immune cells	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			(such as mast	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
				element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				Include assays disclosed in deliger et al., delie 00.1-10 (1720), curien and mann, medicus in Enging 216:362,368 (1002): Hanthorn at al. Proc Natl Acad Sci 118A 85:6342,6346 (1988): De
				Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-
				7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et
				al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
300	1100TO011	0.50	Deschiosion of	illast Cells. A account for maconing a variancian of MAM are well brown in the art and may be used or routinely
525	HSQEO84	ارد ارد	Production of	Assays for measuring expression of v.C. and are well-known in the art and may be used of founding of the artificial of polymential of the invention (including out hodies and accurate of
			VCAM IN	modified to assess the ability of polypeptides of the filterity (likelything antibodies and agonists of

endothelial (such as hun umbilical ve endothelial (HUVEC))	
duction of Endothelial cells, which are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used in ICAM production assays include chas human umbilical vein expression of ICAM (CD54), a intergral membrane protein, can be upregulated by cytokines or other factors, and ICAM expression is important in mediating immune and endothelial cells interactions	ν_ ν
n of retion eatic	E
agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety.	agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K. FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening 204 (1999), the contents of each of which is herein incorporated by reference in its entirety.

				Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
327	HSRFZ57	952	Regulation of transcription through the FAS promoter element in hepatocytes	Assays for the regulation of transcription through the FAS promoter element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis. FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Methods in Enzymol. 216:362–368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays, such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.
328	HSSGD52	953	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol

				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
	HSSGD52	953	Activation of transcription through serum response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., I Immunol
				153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.
329	HSUBW09	954	Regulation of transcription through the FAS promoter element in hepatocytes	Assays for the regulation of transcription through the FAS promoter element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis. FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Biochem J, 317 (Pt 1):257-65 (1996); Berger, et al., Gene 66:1-10 (1988); and, U.S. et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein

such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.	CD152 FMAT. CD152 (a.k.a. CTLA.4) expression is restricted to activated T cells. CD152 is a negative regulator of T cell proliferation. Reduced CD152 expression has been linked to hyperproliferative and autoimmune diseases. Overexpression of CD152 may lead to impaired immunoresponses. Assays for immunomodulatory proteins important in the maintenance of T cell homeostasis and expressed almost exclusively on CD4+ and CD8+ T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate the activation of T cells, maintain T cell homeostasis, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers, such as CD152, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Curr Opin Immunol 11(3):294-300 (1999); and Saito T, Curr Opin Immunol 10(3):313-321 (1998), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.	This reporter assay measures activation of the GATA-3 signaling pathway in FIMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
	Upregulation of CD152 and activation of T cells	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).
	954	955
	HSUBW09	HSVAT68
	329	330

				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
331	HSVBU91	956	Activation of transcription through cAMP response element (CRE) in preadipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Preadipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 373-L1 cells. 373-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 373 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
331	HSVBU91	956	Activation of Hepatocyte ERK Signaling Pathway	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK

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				kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-
				1110 (1998); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature
				410(6824):3/40 (2001); and Cobb MH, Frog Biopnys Moi Biol /1(3-4):4/9-300 (1999); the contents of each of which are herein incorporated by reference in its entirety. Rat liver hepatoma
				cells that may be used according to these assays are publicly available (e.g., through the ATCC).
				Exemplary rat liver hepatoma cells that may be used according to these assays include H4lle cells,
				which are known to respond to glucocorticoids, insulin, or cAMP derivatives.
331	HSVBU91	926	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely
				modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is
				measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is
				upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component
				in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of
. —				insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and
				agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J,
				47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et
				al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52
				(1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of
				each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used
				according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
				generated. Exemplary pancreatic cells that may be used according to these assays include HITT15
				Cells. HTT15 are an adherent epithelial cell line established from Syrian hamster islet cells
_				transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors.
_				The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by
				somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219:
				547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
331	HSVBU91	926	Activation of	Assays for the activation of transcription through the CD28 response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through CD28	(including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T
			response element	cells. Exemplary assays for transcription through the CD28 response element that may be used or
			in immune cells	routinely modified to test CD28-response element activity of polypeptides of the invention
			(such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);

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. —			159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.
HSXEC75	957	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
HSYBG37	958	Activation of Adipocyte ERK Signaling Pathway	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse

				adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
. 333	HSYBG37	958	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirery. Exemplary cells that may be used according to these assays include essinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reactions. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun Mt2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils." Clin Exp Immunol; Oct;122(1):20-7 (2000); Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa Ar phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosyborylation of JUN N-terminal kinase and failure of prednisolone to inhibit number of predni
334	HSZAF47	959	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the

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assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-lun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of predniscolone to inhibit JUN N-terminal	kinase phosphorylation J Allergy Clin Immunol; Sep; 104(3 Ft 1):303-74 (1999); the contents of each of which are herein incorporated by reference in its entirety. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.
	Activation of transcription through NFAT response in immune cells (such as T-cells).
	096
	HTADX17
	335

Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the MOLT4 cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Brazymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the MOLT4, that may be used according to these assays are publicly available (e.g., through the ATCC).	Caspase Apoptosis Rescue. Assays for caspase apoptosis rescue are well known in the art and may be used or routinely modified to assess the ability of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to inhibit caspase protease-mediated apoptosis. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis rescue of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Romeo et al., Cardiovasc Res 45(3): 788-794 (2000); Messmer et al., Br J Pharmacol 127(7): 1633-1640 (1999); and J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays are publicly available (e.g.,
Activation of transcription through GAS response element in immune cells (such as T-cells).	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Protection from Endothelial Cell Apoptosis.
096	096	961
HTADX17	HTADX17	HTAEE28
335	335	336

				through commercial sources). Exemplary endothelial cells that may be used according to these assays include bovine aortic endothelial cells (bAEC), which are an example of endothelial cells which line blood vessels and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.
336	HTAEE28	961	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J. 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
337	нтессо5	962	Regulation of viability and proliferation of pancreatic beta cells.	Assays for the regulation of viability and proliferation of cells in vitro are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate viability and proliferation of pancreatic beta cells. For example, the Cell Titer-Glo luminescent cell viability assay measures the number of viable cells in culture based on quantitation of the ATP present which signals the presence of metabolically active cells. Exemplary assays that may be used or routinely modified to test regulation of viability and proliferation of pancreatic beta cells by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Friedrichsen BN, et al., Mol Endocrinol, 15(1):136-48 (2001); Huotari MA, et al., Endocrinology, 139(4):1494-9 (1998); Hugl SR, et al., J Biol Chem 1998 Jul 10;273(28):17771-9 (1998), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that

may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response	used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the particular and exhibits many characteristics of immature	mast cells. CD154 FMAT. CD154 (a.k.a., CD40L) expression is induced following activation of T cells. Interraction between CD154 and CD40 on B cells is required for correct antibody class switching and germinal center formation. Mutations in CD154 are linked to immunodeficiencies and increased susceptibility to infections. Assays for immunomodulatory proteins important for antibody class switching and TH1 function and expressed on activated T helper lymphocytes are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate the activation of T cells, modulate antibody class switching, mediate TH1 function, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins
	Activation of transcription through GATA-3 response element in immune cells (such as mast		Upregulation of CD154 and activation of T cells
	963		963
	HTEDY42		HTEDY42
	338		. 338

			,	Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204
				al., J Leukoc Biol 63(4):418:428 (1998); and Skov et al., 164(7):3500-3505 (2000), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used
				according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T
				Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.
339	HTEEB42	964	Regulation of	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be
			transcription of	used or routinely modified to assess the ability of polypeptides of the invention (including
			henatocytes	antioodies and agonists of antagonists of the invention) to regulate transcription of ivialic enzyme, a key enzyme in linogenesis. Malic enzyme is involved in linogenesisand its expression is stimulted
			an foundar	by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified
				as putative PPAR response elements. ME promoter may also responds to AP1 and other
				transcription factors. Exemplary assays that may be used or routinely modified to test for regulation
				of transcription of Malic Enzyme (in hepatocytes) by polypeptides of the invention (including
				antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S.,
				et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol,
				8(10):1561-9 (1994); barroso, 1., et al., J Biol Chem, 2/4(25):17997-8004 (1999); Jpenberg, A., et al. TRiol Chem 272/32):20108-20117 (1997): Berger et al. Gene 66:1-10 (1988): and Cullen B.
				et al., Methods in Enzymol. 216:362–368 (1992), the contents of each of which is herein
				incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are
				publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary
				hepatocytes that may be used according to these assays includes the mouse 3T3-L1 cell line. 3T3-
				L1 is a mouse preadipocyte cell line (adherent). It is a continuous substrain of 3T3 fibroblasts
				developed through clonal isolation. Cells undergo a pre-adipocyte to adipose-like conversion under
				appropriate differentiation culture conditions.
340	HTEFU65	965	Activation of	Assays for the activation of transcription through the cAMP response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through cAMP	(including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate
			response element	CREB transcription factors, and modulate expression of genes involved in a wide variety of cell

functions. For example, a 3T3-LI/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CREB binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Preadipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays includes the mouse 373-L1 cell line. 373-L1 is a mouse preadipocyte cell line (adherent). It is a continuous substrain of 373 fibroblasts developed through clonal isolation. Cells undergo a pre-adipocyte to adipose-like conversion under
(CRE) in pre- adipocytes.	Regulation of transcription of Malic Enzyme in hepatocytes
	965
	HTEFU65
	340

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				appropriate differentiation culture conditions.
340	HTEFU65	965	Myoblast cell proliferation	Assays for muscle cell proliferation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit myoblast cell proliferation. Exemplary assays for myoblast cell proliferation that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays disclosed in: Soeta, C., et al. "Possible role for the c-ski gene in the proliferation of myogenic cells in regenerating skeletal muscles of rats" Dev Growth Differ Apr;43(2):155-64 (2001); Ewton DZ, et al., "IGF binding proteins 4, -5 and -6 may play specialized roles during L6 myoblast proliferation and differentiation" J Endocrinol Mar;144(3):539-53 (1995); and, Pampusch MS, et al., "Effect of transforming growth factor beta on proliferation of L6 and embryonic porcine myogenic cells" J Cell Physiol Jun;143(3):524-8 (1990); the contents of each of which are herein incorporated by reference in their entirety. Exemplary myoblast L6 cells are an adherent rat myoblast L6 cell line, isolated from primary cultures of rat thigh muscle, that fuse to form multinucleated myotubes and striated fibers after culture in differentiation media.
340	HTEFU65	965	Production of IFNgamma using a T cells	FNgamma FMAT. IFNg plays a central role in the immune system and is considered to be a proinflammatory cytokine. IFNg promotes TH1 and inhibits TH2 differentiation; promotes IgG2a and inhibits IgE secretion; induces macrophage activation; and increases MHC expression. Assays for immunomodulatory proteins produced by T cells and NK cells that regulate a variety of inflammatory activities and inhibit TH2 helper cell functions are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate humonal or cellmediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as Interferon gamma (IFNg), and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Gonzalez et al., J Clin Lab Anal 8(5):225-233 (1995); Billiau et al., Ann NY Acad Sci 856:22-32 (1998); Boehm et al., Annu Rev Immunol 15:749-795 (1997), and Rheumatology (Oxford) 38(3):214-20 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in

				the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.
340	HTEFU65	965	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
341	HTEG142	996	Activation of transcription through NFAT response in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays

				include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.
342	нтениз1	967	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
343	нтениоз	896	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the

				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
343	нтени93	896	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL-10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
344	HTEIP36	696	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the MOLT4, that may be used according to these assays are publicly available (e.g., through the ATCC).
344	нтвР36	696	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to

modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL-10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL-10, IL-13, IL-5 and IL-6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Assays for the regulation of transcription through the PEPCK promoter are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the PEPCK promoter in a reporter construct and regulate liver gluconeogenesis. Exemplary assays for regulation of transcription through the PEPCK promoter that may be used or routinely modified to test for PEPCK promoter activity (in hepatocytes) of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Lochhead et al., Diabetes 49(6):896-903 (2000); and Yeagley et al., J Biol Chem 275(23):17814-17820 (2000), the contents of each of which is herein incorporated by reference in its entirety. Hepatocyte cells that may be used according to these assays include H4lle cells, which contain a tyrosine amino transferase that is inducible with glucocorticoids, insulin, or cAMP derivatives.	Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to measure influx of calcium. Cells normally have very low concentrations of cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin L.S, et al., Endocrinology, 136(10):4589-601 (1995);Mogami H, et
	Regulation of transcription through the PEPCK promoter in hepatocytes	Stimulation of Calcium Flux in pancreatic beta cells.
	970	970
	HTELP17	HTELP17
	345	345

				al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551: Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
346	HTELS08	971	Regulation of transcription through the PEPCK promoter in hepatocytes	Assays for the regulation of transcription through the PEPCK promoter are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the PEPCK promoter in a reporter construct and regulate liver gluconeogenesis. Exemplary assays for regulation of transcription through the PEPCK promoter that may be used or routinely modified to test for PEPCK promoter activity (in hepatocytes) of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Lochhead et al., Diabetes 49(6):896-903 (2000); and Yeagley et al., J Biol Chem 275(23):17814-17820 (2000), the contents of each of which is herein incorporated by reference in its entirety. Hepatocyte cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary liver hepatoma cells that may be used according to these assays include H4lle cells, which contain a tyrosine amino transferase that is inducible with glucocorticoids, insulin, or cAMP derivatives.
347	HTEPG70	972	Activation of transcription through cAMP response element (CRE) in preadipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of

		
polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Preadipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. Pre-adipocytes that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the
	Activation of transcription through serum response element in pre-adipocytes.	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).
	972	972
·	HTEPG70	HTEPG70
	347	347

				invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 83:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
347	HTEPG70	972	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Tumer et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
347	HTEPG70	972	Activation of transcription through NFKB response element	This reporter assay measures activation of the NFkB signaling pathway in Ku812 human basophil cell line. Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB

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			in immune cells (such as basophils).	transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Marone et al, Int Arch Allergy Immunol 114(3):207-17 (1997), the contents of each of which are herein incorporated by reference in its entirety. Basophils that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human basophil cell lines that may be used according to these assays include Ku812, originally established from a patient with chronic myelogenous leukemia. It is an immature prebasophilic cell line that can be induced to differentiate into mature basophils.
347	HTEPG70	972	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-85:6342-6346 (1988); black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-85:6342-6346 (1988); black et al., Virus Gnes assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
347	HTEPG70	972	Activation of transcription through serum response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each

				of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.
348	HTHCA18	973	Production of GM-CSF	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage. Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory cytokine. Assays for immunomodulatory proteins that promote the production of GM-CSF are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF, and that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach." Chapter 6:138-160 (2000); and Ye et al., J Leukoc Biol (82(2):225-233, the contents of each of which are herein ancorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclosed herein or otherwise known in the at. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor cells and also recognize antibody bound on target cells, via NK Fc receptors, leading to cell-mediated cytotoxicity.
349	нтлма95	974	Activation of transcription through cAMP response element (CRE) in preadipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of

polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923 (1998), the contents of each of which are herein incorporated by reference in its entirety. Preadipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of altergic reactions, they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogenactivated protein kinase in human eosinophils." Clin Exp Immunol, Oct;122(1):20-7 (2000); Hebestreet H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils. The Contriorstence of procholyation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation. J Allerey Clin Immunolis ason associated with enhanced
	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).
	974
	HTJMA95
	349

				each of which are herein incorporated by reference in its entirety.
349	HTJMA95	974	Activation of	Assays for the activation of transcription through the AP1 response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through AP1	(including antibodies and agonists or antagonists of the invention) to modulate growth and other cell
			response element	functions. Exemplary assays for transcription through the API response element that may be used
			in immune cells	or routinely modified to test API-response element activity of polypeptides of the invention
			(such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem
				272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al.,
				Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by
				reference in its entirety. Human T cells that may be used according to these assays are publicly
				available (e.g., through the ATCC). Exemplary human T cells that may be used according to these
				assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.
349	HTJMA95	974	Activation of	Assays for the activation of transcription through the CD28 response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through CD28	(including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T
			response element	cells. Exemplary assays for transcription through the CD28 response element that may be used or
			in immune cells	routinely modified to test CD28-response element activity of polypeptides of the invention
			(such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol
				159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J
				Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference
				in its entirety. T cells that may be used according to these assays are publicly available (e.g.,
				through the ATCC). Exemplary human T cells that may be used according to these assays include
				the SUPT cell line, which is a suspension culture of L-2 and L-4 responsive T cells.
349	HTJMA95	974	Activation of	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			transcription	response element are well-known in the art and may be used or routinely modified to assess the
			through NFAT	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			response element	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			in immune cells	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
			(such as T-cells).	element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)

				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
349	HTJMA95	974	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.
349	HTJMA95	974	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL 10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL 10, IL 13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and

asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage. Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory cytokine. Assays for immunomodulatory proteins that promote the production of GM-CSF are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and modulate the growth and differentiation of leukocytes. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach." Chapter 6:138-160 (2000); and Ye et al., J Leukoc Biol (58(2):225-233, the contents of each of which are herein incorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC) or may be isolated using techniques disclosed herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor pale and also recognize antihody bound on target cells was waitedy-independent killing of tells show antibody-independent killing of tells of the cells and also recognize antihody of tells show antibody-independent killing of tells of the cells and	mediated cytotoxicity. This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold
	Production of GM-CSF	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).
	975	976
	HTJML75	HTLAA40
	350	351

				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
352	HTLEP53	77.6	Endothelial Cell Apoptosis	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Induction of apoptosis in endothelial cells supporting the vasculature of tumors is associated with tumor regression due to loss of tumor blood supply. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these assays include bovine aortic endothelial cells (bAEC), which are an example of endothelial cells which line blood vessels and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.
352	HTLEP53	977	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of

each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).	Assays for the activation of transcription through the Signal Transducers and Activators of Transcription (STAT6) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT6 transcription factors and modulate the expression of multiple genes. Exemplary assays for transcription through the STAT6 response element that may be used or routinely modified to test STAT6 response element activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Georas et al., Blood 92(12):4529-4538 (1998); Moffatt et al., Transplantation 69(7):1521-1523 (2000); Curiel et al., Eur J Immunol 27(8):1982-1987 (1997); and Masuda et al., J Biol Chem 275(38):29331-29337 (2000), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary rat natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the
	Production of ICAM-1	Activation of transcription through STAT6 response element in immune cells (such as natural killer cells).	Activation of transcription
	978	979	626
	HTLFE57	HTLIV19	HTLIV19
·	353	354	354

354 ·	HTLIV19	979	through NFAT response element in immune cells (such as natural killer cells). Activation of transcription through serum response element in immune cells (such as natural killer cells). Production of ICAM-1	ability of polypeptides of the invention (including antibodies and agonists or antiagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Braymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Aranburu et al., Exp Med 182(3):801-810 (1995); De Boer et al., int J Biochem Cell Biol 31(10):121-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J 11(10):121-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J 810 (1992); Heathorn et al., Fazer et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J 11(10):121-1236 (1999); Araser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J 810 (109):1438-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. NK cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human NK cells that may be used acrotining modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of excerding to these assays are publicly available (e.g., through the ATCC). Exemplary and agonists or the invention to including antibo
				Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365

			(2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include Aortic Smooth Muscle Cells (AOSMC); such as bovine AOSMC.	
HTODK73	981	Activation of transcription through NFAT response in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.	
нтонр42	982	Production of IL- 10 and activation of T-cells.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions	

-				using peripheral blood lymphocytes isolated from cord blood.
358	HTOHIMIS	983	Activation of JNK	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation,
			Signaling Pathway	or apoptosis are well known in the art and may be used or routinely modified to assess the ability of
			in immune cells	polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to
			(such as	promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase
			eosinophils).	activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides
- <u>-</u> -				of the invention (including antibodies and agonists or antagonists of the invention) include the
				assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res
				247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin,
				Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the
				contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that
_				may be used according to these assays include eosinophils. Bosinophils are important in the late
				stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of
				late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to
				assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists
				of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in
				eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in
				dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-
				activated protein kinase in human eosinophils" Clin Exp Immunol; Oct;122(1):20-7 (2000);
				Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med;
				Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR,
_				et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced
				phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal
				kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of
				each of which are herein incorporated by reference in its entirety.
359	HTPBW79	984	Activation of	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell
			transcription	line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production.
			through NFAT	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			response element	response element are well-known in the art and may be used or routinely modified to assess the
			in immune cells	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			(such as mast	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
<u>. </u>			cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
				element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)

				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 210:302-308 (1992); Henthom et al., 170c Natt Acad Sci USA 83:0342-0340 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-
				7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incomorated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
359	HTPBW79	984	Activation of	Assays for the activation of transcription through the API response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through AP1	(including antibodies and agonists or antagonists of the invention) to modulate growth and other cell
			response element	functions. Exemplary assays for transcription through the AP1 response element that may be used
			in immune cells	or routinely modified to test AP1-response element activity of polypeptides of the invention
			(such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1988); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Rellahan et al., J Biol Chem
			,	272(49):30806-30811 (1997); Chang et al., Mol Cell Biol 18(9):4986-4993 (1998); and Fraser et al.,
				Eur J Immunol 29(3):838-844 (1999), the contents of each of which are herein incorporated by
				reference in its entirety. Human T cells that may be used according to these assays are publicly
				available (e.g., through the ATCC). Exemplary human T cells that may be used according to these
				assays include the SUPT cell line, which is an IL-2 and IL-4 responsive suspension-culture cell line.
329	HTPBW79	984	Activation of	Assays for the activation of transcription through the CD28 response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through CD28	(including antibodies and agonists or antagonists of the invention) to stimulate IL-2 expression in T
		_	response element	cells. Exemplary assays for transcription through the CD28 response element that may be used or
			in immune cells	routinely modified to test CD28-response element activity of polypeptides of the invention
			such as T-cells).	(including antibodies and agonists or antagonists of the invention) include assays disclosed in
				Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992);
				Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); McGuire and Iacobelli, J Immunol
				159(3):1319-1327 (1997); Parra et al., J Immunol 166(4):2437-2443 (2001); and Butscher et al., J
				Biol Chem 3(1):552-560 (1998), the contents of each of which are herein incorporated by reference
				in its entirety. T cells that may be used according to these assays are publicly available (e.g.,

2	(NFAT) sss the of the lved in ponse if ention) ls in 988); Cell Biol n et al., J orated by vailable assays ells.	nown in the nation scription iscription iscription ists or sit USA (3):838-ety. TATCC).	sly gonists or used to
nd IL-4 responsive T cells.	ear Factor of Activated T cell ed or routinely modified to as ies and agonists or antagonist fullate expression of genes invertipation through the NFAT reAT-response element activity onists or antagonists of the in 198); Cullen and Malm, Methacad Sci USA 85:6342-6346 (); De Boer et al., Int J Biocher (3):838-844 (1999); and Yese (3):838-844 (1999); and Yese ach of which are herein incorping to these assays are publicly may be used according to these of the second of the sec	'B response element are well-lility of polypeptides of the invention) to regulate NFKB transes. Exemplary assays for transers including antibodies and aggierger et al., Gene 66:1-10 (19 nthom et al., Proc Natl Acad S 117 (1997); and Fraser et al., porated by reference in its ent sly available (e.g., through the hese assays include the SUPT T cells.	e art and may be used or routintion (including antibodies and imple, the FLPR assay may be
through the ATCC). Exemplarly furnian 1 cens that may be used according to these assays include the SUPT cell line, which is a suspension culture of $L-2$ and $L-4$ responsive Γ cells.	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the SUPT cell line, which is a suspension culture of IL-2 and IL-4 responsive T cells.	Assays for measuring calcium flux are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mobilize calcium. For example, the FLPR assay may be used to
the SUPT cell line, wh	Assays for the activati response element are vability of polypeptides invention) to regulate immunomodulatory fuelement that may be upolypeptides of the invinclude assays discloss Enzymol 216:362-368 Serfling et al., Biochir 31(10):1221-1236 (19 Biol Chem 268(19):14 reference in its entiret (e.g., through the ATC include the SUPT cell	Assays for the activati art and may be used on (including antibodies a factors and modulate et through the NFKB res response element activantagonists of the inve and Malm, Methods it 85:6342-6346 (1988); 844 (1999), the conter cells that may be used Exemplary human T c which is a suspension	Assays for measuring modified to assess the antagonists of the inve
	Activation of transcription through NFAT response element in immune cells (such as T-cells).	Activation of transcription through NFKB response element in immune cells (such as T-cells).	Stimulation of Calcium Flux in pancreatic beta
	984	984	985
-	HTPBW79	HTPBW79	HTPCS72
	359	359	360

		·		compared to much higher extracellular calcium. Extracellular factors can cause an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Satin LS, et al., Endocrinology, 136(10):4589-601 (1995); Mogami H, et al., Endocrinology, 136(7):2960-6 (1995); Richardson SB, et al., Biochem J, 288 (Pt 3):847-51 (1992); and, Meats, JE, et al., Cell Calcium 1989 Nov-Dec;10(8):535-41 (1989), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HTTT15 Cells. HTTT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219:
361	нтрін83	986	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999); the contents of according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219:

362	HTSEW 17	987	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulinome. References:
362	HTSEW17	987	Activation of transcription through NFKB response element in immune cells (such as B-cells).	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Gri G, et al., Biol Chem, 273(11):6431-6438 (1998); Pyatt DW, et al., Cell Biol Toxicol 2000;16(1):41-51 (2000); Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of which are herein incorporated by reference in its entirety. Immune cells that may be used according to these assays include the Reh B-cell line.
363	HTTBI76	886	Stimulation of insulin secretion from pancreatic	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is

			beta cells.	measured by FMAT using anti-rat insulin antihodies. Insulin secretion from pancreatic heta cells is
				upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component
				in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of
				insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and
				277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al.,
				FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-
				204 (1999), the contents of each of which is herein incorporated by reference in its entirety.
				Pancreatic cells that may be used according to these assays are publicly available (e.g., through the
				ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according
				to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from
				cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics
				typical of native pancreatic beta cells including glucose inducible insulin secretion. References:
				Asfari et al. Endocrinology 1992 130:167.
363	HTTBI76	886	Upregulation of	CD69 FMAT. CD69 is an activation marker that is expressed on activated T cells, B cells, and NK
			CD69 and	cells. CD69 is not expressed on resting T cells, B cells, or NK cells. CD69 has been found to be
			activation of T	associated with inflammation. Assays for immunomodulatory proteins expressed in T cells, B cells,
			cells	and leukocytes are well known in the art and may be used or routinely modified to assess the ability
				of polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				to modulate the activation of T cells, and/or mediate humoral or cell-mediated immunity.
				Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface
				markers, such as CD69, and the activation of T cells. Such assays that may be used or routinely
				modified to test immunomodulatory activity of polypeptides of the invention (including antibodies
				and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia
				et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical
				approach" Chapter 6:138-160 (2000); Ferenczi et al., J Autoimmun 14(1):63-78 (200); Werfel et
				al., Allergy 52(4):465-469 (1997); Taylor-Fishwick and Siegel, Eur J Immunol 25(12):3215-3221
				(1995); and Afetra et al., Ann Rheum Dis 52(6):457-460 (1993), the contents of each of which are
				herein incorporated by reference in its entirety. Human T cells that may be used according to these
				assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T
				cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and
				CD3, CD4, or CD8. These cells mediate humoral or cell-mediated immunity and may be
				preactivated to enhance responsiveness to immunomodulatory factors.
364	HTTBS64	686	Regulation of	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be

			transcription of	used or routinely modified to assess the ability of polypeptides of the invention (including
			Malic Enzyme in	antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a
			neparocytes	by insulin. ME promoter contains two direct repeat (DR1)- like elements. MEp and MEd identified
				as putative PPAR response elements. ME promoter may also responds to AP1 and other
				transcription factors. Exemplary assays that may be used or routinely modified to test for regulation
				of transcription of Malic Enzyme (in hepatocytes) by polypeptides of the invention (including
				antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S.,
				et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol,
				8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et
				al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B.,
				et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein
			-	incorporated by reference in its entirety. Hepatocytes that may be used according to these assays are
				publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary
				hepatocytes that may be used according to these assays includes the mouse 3T3-L1 cell line. 3T3-
				Li is a mouse preadipocyte cell line (adherent). It is a continuous substrain of 3T3 fibroblasts
				developed through clonal isolation. Cells undergo a pre-adipocyte to adipose-like conversion under
				appropriate differentiation culture conditions.
365	HTWCT03	066	Regulation of	Assays for the regulation (i.e. increases or decreases) of viability and proliferation of cells in vitro
			viability or	are well-known in the art and may be used or routinely modified to assess the ability of polypeptides
			proliferation of	of the invention (including antibodies and agonists or antagonists of the invention) to regulate
			immune cells	viability and proliferation of eosinophil cells and cell lines. For example, the CellTiter-Gloô
			(such as human	Luminescent Cell Viability Assay (Promega Corp., Madison, WI, USA) can be used to measure the
			eosinophil EOL-1	number of viable cells in culture based on quantitation of the ATP present which signals the
			cells).	presence of metabolically active cells. Eosinophils are a type of immune cell important in allergic
	-			responses; they are recruited to tissues and mediate the inflammtory response of late stage allergic
				reaction. Eosinophil cell lines that may be used according to these assays are publicly available
				and/or may be routinely generated. Exemplary eosinophil cells that may be used according to these
				assays include EOL-1 Cells.
365	HTWCT03	066	Production of	TNFa FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells,
			TNF alpha by	fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and
			dendritic cells	cytotoxic effects on a variety of cells are well known in the art and may be used or routinely
				modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to mediate immunomodulation, modulate inflammation and

				cytokines such as tumor necrosis factor alpha (TNFa), and the induction or inhibition of an inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Verhasselt et al., Eur J Immunol 28(11):3886-3890 (1198); Dahlen et al., J Immunol 160(7):3585-3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol 65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell	el ii s ç s
365	HTWCF03		Production of IL-8 by immune cells (such as the human EOL-1 eosinophil cells)	Assay that measures the production of the chemokine interleukin-8 (IL-8) from immune cells (such as the EOL-1 human eosinophil cell line) are well known in the art (for example, measurement of IL-8 production by FMAT) and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit. Eosinophils are a type of immune cell important in allergic responses; they are recruited to tissues and mediate the inflammtory response of late stage allergic reaction. IL8 is a strong immunomodulator and may have a potential proinflammatory role in immunological diseases and disorders (such as allergy and asthma).	S S
365	HTWCT03	066	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol	o be

				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
365	HTWCT03	066	Activation of transcription through NFAT response element in immune cells (such as mast cells).	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998); the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
365	HTWCT03	066	Production of VCAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to meaure the upregulation of cell surface VCAM-I expression in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors,

			and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels: thus VCAM expression plays a role in promoting immune and inflammatory responses.
HTWCT03	066	Production of ICAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Endothelial cells, which are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used in ICAM production assays include human umbilical vein endothelial cells (HUVEC), and are available from commercial sources. The expression of ICAM (CD54),a intergral membrane protein, can be upregulated by cytokines or other factors, and ICAM expression is important in mediating immune and endothelial cell interactions leading to immune and inflammatory responses. Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365 (1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163 (2000), the contents of each of which is herein incorporated by reference in its entirety.
HTXDW56	991	Activation of transcription through NFAT response in immune cells (such as T-cells).	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Serfling et al., Biochim Biophys Acta 1498(1):1-18 (2000); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Fraser et al., Eur J Immunol 29(3):838-844 (1999); and Yeseen et al., J Biol Chem 268(19):14285-14293 (1993), the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human T cells that may be used according to these assays include the JURKAT cell line, which is a suspension culture of leukemia cells that produce IL-2 when stimulated.
HTXDW56	991	Activation of transcription	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the

			through GAS response element	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide
			in immune cells	variety of cell functions. Exemplary assays for transcription through the GAS response element that
			(sucil as 1 cells).	inay be used of fourthern incomined to test of a response crement with the properties of the invention (including antibodies and agonists or antagonists of the invention) include assays
				disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-
				368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al.,
				Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the
				contents of each of which are herein incorporated by reference in its entirety. Exemplary human T
				cells, such as the MOLT4 cell line, that may be used according to these assays are publicly available
				(e.g., through the ATCC).
366	HTXDW56	991	Activation of	Assays for the activation of transcription through the NFKB response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through NFKB	(including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription
_			response element	factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription
			in immune cells	through the NFKB response element that may be used or rountinely modified to test NFKB-
			(such as T-cells).	response element activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include assays disclosed in Berger et al., Gene 66.1-10 (1998); Cullen
				and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA
				85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-
				844 (1999), the contents of each of which are herein incorporated by reference in its entirety.
				Exemplary human T cells, such as the MOLT4, that may be used according to these assays are
				publicly available (e.g., through the ATCC).
366	HTXDW56	991	Activation of	Assays for the activation of transcription through the NFKB response element are well-known in the
			transcription	art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through NFKB	(including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription
			response element	factors and modulate expression of neuronal genes. Exemplary assays for transcription through the
		-	in neuronal cells	NFKB response element that may be used or routinely modified to test NFKB-response element
			(such as SKNMC	activity of polypeptides of the invention (including antibodies and agonists or antagonists of the
			cells).	invention) include assays disclosed in: Gill JS, et al., Neurobiol Dis, 7(4):448-461 (2000); Tamatani
				M, et al., J Biol Chem, 274(13):8531-8538 (1999); Berger et al., Gene 66:1-10 (1998); Cullen and .
				Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA
				85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J
				Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of

				which are herein incorporated by reference in its entirety. Neuronal cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary neuronal cells that may be used according to these assays include the SKNMC neuronal cell line.
366	HTXDW56	166	Activation of transcription through GAS response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Gamma Interferon Activation Site (GAS) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate STAT transcription factors and modulate gene expression involved in a wide variety of cell functions. Exemplary assays for transcription through the GAS response element that may be used or routinely modified to test GAS-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Matikainen et al., Blood 93(6):1980-1991 (1999); and Henttinen et al., J Immunol 155(10):4582-4587 (1995), the contents of each of which are herein incorporated by reference in its entirety. Exemplary human T cells, such as the SUPT cell line, that may be used according to these assays are publicly available (e.g., through the ATCC).
367	HTXJM03		Regulation of transcription of Malic Enzyme in hepatocytes	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 8(10):1361-9 (1994); Barroso, I., et al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays includes the mouse 373-L1 cell line. 373-L1 is a mouse preadipocyte cell line (adherent). It is a continuous substrain of 373 fibroblasts developed through clonal isolation. Cells undergo a pre-adipocyte to adipose-like conversion under

				annronriate differentiation culture conditions
368	HTXON32	993	Insulin Secretion	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention. For example, insulin secretion is antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Shimizu, H., et al., Endocr J, 47(3):261-9 (2000); Salapatek, A.M., et al., Mol Endocrinol, 13(8):1305-17 (1999); Filipsson, K., et al., Ann N Y Acad Sci, 865:441-4 (1998); Olson, L.K., et al., J Biol Chem, 271(28):16544-52 (1996); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely
				generated. Exemplary pancreatic cells that may be used according to these assays include HITT15 Cells. HITT15 are an adherent epithelial cell line established from Syrian hamster islet cells transformed with SV40. These cells express glucagon, somatostatin, and glucocorticoid receptors. The cells secrete insulin, which is stimulated by glucose and glucagon and suppressed by somatostatin or glucocorticoids. ATTC# CRL-1777 Refs: Lord and Ashcroft. Biochem. J. 219: 547-551; Santerre et al. Proc. Natl. Acad. Sci. USA 78: 4339-4343, 1981.
369	HUDBZ89	994	Activation of transcription through cAMP response element (CRE) in preadipocytes.	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP, regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. For example, a 3T3-L1/CRE reporter assay may be used to identify factors that activate the cAMP signaling pathway. CREB plays a major role in adipogenesis, and is involved in differentiation into adipocytes. CRE contains the binding sequence for the transcription factor CREB (CRE binding protein). Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Reusch et al., Mol Cell Biol 20(3):1008-1020 (2000); and Klemm et al., J Biol Chem 273:917-923

			-	adipocytes that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
369	HUDBZ89	994	Production of GM-CSF	GM-CSF FMAT. GM-CSF is expressed by activated T cells, macrophages, endothelial cells, and fibroblasts. GM-CSF regulates differentiation and proliferation of granulocytes- macrophage progenitors and enhances antimicrobial activity in neutrophils, monocytes and macrophage. Additionally, GM-CSF plays an important role in the differentiation of dendritic cells and monocytes, and increases antigen presentation. GM-CSF is considered to be a proinflammatory cytokine. Assays for immunomodulatory proteins that promote the production of GM-CSF are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to mediate immunomodulation and modulate the growth and differentiation of leukocytes. Exemplary assays that test for immunomodulatory proteins evaluate the production of cytokines, such as GM-CSF, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); and Y et al., J Leukoc Biol (58(2):225-233; the contents of each of which are herein incorporated by reference in its entirety. Natural killer cells that may be used according to these assays are publicly available (e.g., through the ATCC) or may be isolated using techniques alsolated herein or otherwise known in the art. Natural killer (NK) cells are large granular lymphocytes that have cytotoxic activity but do bind antigen. NK cells show antibody-independent killing of tumor cells and also recognize antibody bound on target cells, via NK Fc receptors, leading to cell-
370	HUFBY15	995	Activation of T- Cell p38 or JNK Signaling Pathway.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem

:				379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
371	HUFCJ30	966	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
372	HUKAH51	997	Protection from Endothelial Cell Apoptosis.	Caspase Apoptosis Rescue. Assays for caspase apoptosis rescue are well known in the art and may be used or routinely modified to assess the ability of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to inhibit caspase protease-mediated apoptosis. Exemplary assays for caspase apoptosis that may be used or routinely modified to test caspase apoptosis rescue of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Romeo et al., Cardiovasc Res 45(3): 788-794 (2000); Messmer et al., Br J Pharmacol 127(7): 1633-1640 (1999); and J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Endothelial cells that may be used according to these essays are publicly available (e.g., through commercial sources). Exemplary endothelial cells that may be used according to these

				assays include bovine aortic endothelial cells (bAEC), which are an example of endothelial cells which line blood vessels and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation.
372	HUKAHSI	997	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions, they are recruited to tissues and mediate the inflammatory response of late stage allergic reaction. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in dexamethasone-induced apoptosis and activation of cluding antibodies and agonists or antagonists of the invention of manane activation of as receptor signaling by nitric oxide in eosinophils. I Exp Med; Feb 2;187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, et al., "In vivo resistance to corticosteroids in bronchial asthma as associated with enhanced phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal kinase phosphorylation of JUN N-terminal sits entirety.
373	HUKBT29	866	Production of IL6 by primary human aortic smooth muscle or normal human dermal fibroblast cells (without or with	Assay to measure regulation of production of Interleukin-6 (IL-6) by either human aortic smooth muscle cells or normal human dermal fibroblasts minus or plus costimulation with TNFalpha (TNFa). Human aortic smooth muscle cells or normal human dermal fibroblasts may be obtained from commercial sources; these cells are important structural and functional components of blood vessels and connective tissue, respectiviely. Interleukin-6 (IL-6) is a key molecule in chronic inflammation and has been implicated in the progression of atherosclerosis, stroke, arthritis and other vascular and inflammatory diseases. Deregulated expression of IL-6 has been linked to

			Continuitation with	priest manuary disperses also many actions and action and a bearing bearing the manual formation of the second
			TNFalpha).	autominimo disease, prasinacytomas, inyciomas, and cinomic righer promieranye diseases. Assays for immunomodulatory and differentiation factor proteins produced by a large variety of cells where
				the expression level is strongly regulated by cytokines, growth factors, and hormones are well
				known in the art and may be used or routinely modified to assess the ability of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) to mediate immunomedulation and production of IL-6.
373	HUKBT29	866	Production of	TNFa FMAT. Assays for immunomodulatory proteins produced by activated macrophages, T cells,
			TNF alpha by	fibroblasts, smooth muscle, and other cell types that exert a wide variety of inflammatory and
			dendritic cells	cytotoxic effects on a variety of cells are well known in the art and may be used or routinely
				modified to assess the ability of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) to mediate immunomodulation, modulate inflammation and
				cytotoxicity. Exemplary assays that test for immunomodulatory proteins evaluate the production of
				cytokines such as tumor necrosis factor alpha (TNFa), and the induction or inhibition of an
				inflammatory or cytotoxic response. Such assays that may be used or routinely modified to test
				immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include assays disclosed in Miraglia et al., J Biomolecular Screening
				4.193-204(1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000);
				Verhasselt et al., Eur J Immunol 28(11):3886-3890 (1198); Dahlen et al., J Immunol 160(7):3585-
				3593 (1998); Verhasselt et al., J Immunol 158:2919-2925 (1997); and Nardelli et al., J Leukoc Biol
				65:822-828 (1999), the contents of each of which are herein incorporated by reference in its entirety.
				Human dendritic cells that may be used according to these assays may be isolated using techniques
				disclosed herein or otherwise known in the art. Human dendritic cells are antigen presenting cells in
				suspension culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell
				proliferation and functional activities.
373	HUKBT29	866	Production of IL-4	IL 4 FMAT. Assays for immunomodulatory proteins secreted by TH2 cells that stimulate B cells, T
				cells, macrophages and mast cells and promote polarization of CD4+ cells into TH2 cells are well
				known in the art and may be used or routinely modified to assess the ability of polypeptides of the
				invention (including antibodies and agonists or antagonists of the invention) to mediate
				immunomodulation, stimulate immune cells, modulate immune cell polarization, and/or mediate
				humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins
				evaluate the production of cytokines, such as IL-4, and the stimulation of immune cells, such as B
				cells, T cells, macrophages and mast cells. Such assays that may be used or routinely modified to
				test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists
				or antagonists of the invention) include the assays disclosed in Miraglia et al., J Biomolecular

				incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTL cell line, which is an IL-2 dependent suspension-culture cell line.	ng e
				with cytotoxic activity.	
374	HUSXS50	666	Activation of	Assays for the activation of transcription through the NFKB response element are well-known in the	the
			transcription	art and may be used of rounnely modified to assess the ability of polypeptides of the invention (including partial descripts or antigonists of the invention) to requise NECP transcription	
			response element	(ilicituting antibodies and agoinsts of antagoinsts of the invention) to regulate 141 AD dailscription factors and modulate expression of imminomodulatory genes. Exemplary assays for transcription	 :
				through the NFKB response element that may be used or rountinely modified to test NFKB-	
			as E(response element activity of polypeptides of the invention (including antibodies and agonists or	
			cells).	antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen	en
				and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA	
				85:6342-6346 (1988); Valle Blazquez et al, Immunology 90(3):455-460 (1997); Aramburau et al., J	
				Exp Med 82(3):801-810 (1995); and Fraser et al., 29(3):838-844 (1999), the contents of each of	
				which are herein incorporated by reference in its entirety. For example, a reporter assay (which	
				measures increases in transcription inducible from a NFkB responsive element in EOL-1 cells) may	lay
				link the NFKB element to a repeorter gene and binds to the NFKB transcription factor, which is	-
				upregulated by cytokines and other factors. Exemplary immune cells that may be used according to	<u>유</u>
				these assays include eosinophils such as the human EOL-1 cell line of eosinophils. Eosinophils are	are
				a type of immune cell important in the allergic responses; they are recruited to tissues and mediate	و
				the inflammtory response of late stage allergic reaction. Eol-1 is a human eosinophil cell line.	
374	HUSXS50	666	Calcium flux in	Assays for measuring calcium flux are well-known in the art and may be used or routinely modified	jed
			immune cells	to assess the ability of polypeptides of the invention (including antibodies and agonists or	
			(such as	antagonists of the invention) to mobilize calcium. Cells normally have very low concentrations of	Jo Jo
			monocytes)	cytosolic calcium compared to much higher extracellular calcium. Extracellular factors can cause	စ္
				an influx of calcium, leading to activation of calcium responsive signaling pathways and alterations	Suc
				in cell functions. Exemplary assays that may be used or routinely modified to measure calcium flux	flux
				in immune cells (such as monocytes) include assays disclosed in: Chan, CC, et al., J Pharmacol Exp	Exp
				Ther, 269(3):891-896 (1994); Andersson, K, et al., Cytokine, 12(12):1784-1787 (2000); Scully, SP,	SP,
				et al., J Clin Invest, 74(2) 589-599 (1984); and, Sullivan, E, et al., Methods Mol Biol, 114:125-133	33
				(1999), the contents of each of which is herein incorporated by reference in its entirety. Cells that	=
				may be used according to these assays are publicly available (e.g., through the ATCC) and/or may	<u>~</u>
				be routinely generated. Exemplary cells that may be used according to these assays include the	
				THP-1 monocyte cell line.	

HUVEB53 1000 Regulation of Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or apoptosis in agonists or analogonists of the invention) to promote caspase protease-meditated apoptosis. Apoptosis in agonists or analogonist of the fivention) to promote caspase protease-meditated apoptosis. Apoptosis in parcreatic beat is associated with induction and progression of diabetees. Exemplizing appropriates of the invention fineluding antibodies and agonists or analogonist activity of polypeptides of the invention (including antibodies and agonists or analogonist paracreatic beat as a social and agonists or an agonists or analogonist than may be used or routinely modified to test capase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antigonist activity of polypeptides of the invention (including antibodies and agonists or antigonist activity of polypeptides of the invention (including antibodies and agonists or antigonist activity of polypeptides of the invention (including antibodies and agonists of the invention) include the assays discloded in Loward A.C. et al., Thinmunol. 166(7):283-6 (1995); Sani, K.S. et al., FBBS Lett, 459(2):283-43 (1999); Zang, S. et al., FBBS Lett, 459(2):283-43 (1999); Zang, S. et al., FBBS Lett, 459(2):283-43 (1999); Latter of the contents of each of which are herein incorporated by reference in its entirely. Pancreatic cells that contents of each of which are herein incorporated by reference in its entirely. Pancreadic cells that may be used according to these assays are publicly available. Pancreadic cells that may be used according to these assays are publicly antible of exposite and according to these assays are publicly and possibly glucagon. ATTC: Addorn may be routinely generated. Exemplary pancreatic cells that may be used according to these assays for the activition induced translational and possibly glucagon. ATTC: ACRL-2057 Chick et al., Thursh may be used according to these assays for transcript		
HUVEB53 1000 HVARW53 1001	Caspase Apoptosis. Assays for caspase apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote caspase protease-mediated apoptosis. Apoptosis in pancreatic beta is associated with induction and progression of diabetes. Exemplary assays for caspase apoptosis that may be used or routinely modified to test capase apoptosis activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in: Loweth, AC, et al., FEBS Lett, 400(3):285-8 (1997); Saini, KS, et al., Biochem Mol Biol Int, 39(6):1229-36 (1996); Krautheim, A., et al., Br J Pharmacol, 129(4):687-94 (2000); Chandra J, et al., Diabetes, 50 Suppl 1:S44-7 (2001); Suk K, et al., J Immunol, 166(7):4481-9 (2001); Tejedo J, et al., FEBS Lett, 459(2):238-43 (1999); Zhang, S., et al., FEBS Lett, 455(3):315-20 (1999); Lee et al., FEBS Lett 485(2-3): 122-126 (2000); Nor et al., J Vasc Res 37(3): 209-218 (2000); and Karsan and Harlan, J Atheroscler Thromb 3(2): 75-80 (1996); the contents of each of which are herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include RIN-m. RIN-m is a rat adherent pancreatic beta cell insulinoma cell line derived from a radiation induced transplantable rat islet cell tumor. The cells produce and secrete islet polypeptide hormones, and produce insulin, somatostatin, and possibly glucagon. ATTC: #CRL-2057 Chick et al. Proc. Natl. Acad. Sci. 1977 74:628; AF et al. Proc. Natl. Acad. Sci. 1977 74:628;	This reporter assay measures activation of the NFAT signaling pathway in HIMC-1 human mast cell line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT) response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate NFAT transcription factors and modulate expression of genes involved in immunomodulatory functions. Exemplary assays for transcription through the NFAT response element that may be used or routinely modified to test NFAT-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available
HUVEB53	Regulation of apoptosis in pancreatic beta cells.	Activation of transcription through NFAT response element in immune cells (such as mast cells).
	0000	1001
376	HUVEB53	HVARW53
	375	376

				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
377	HWAAD63	1002	Regulation of transcription through the FAS promoter element in hepatocytes	Assays for the regulation of transcription through the FAS promoter element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to activate the FAS promoter element in a reporter construct and to regulate transcription of FAS, a key enzyme for lipogenesis. FAS promoter is regulated by many transcription factors including SREBP. Insulin increases FAS gene transcription in livers of diabetic mice. This stimulation of transcription is also somewhat glucose dependent. Exemplary assays that may be used or routinely modified to test for FAS promoter element activity (in hepatocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Xiong, S., et al., Proc Natl Acad Sci U.S.A., 97(8):3948-53 (2000); Roder, K., et al., Eur J Biochem, 260(3):743-51 (1999); Oskouian B, et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays, such as H4IIE cells, are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary hepatocytes that may be used according to these assays include rat liver hepatoma cell line(s) inducible with glucocorticoids, insulin, or cAMP derivatives.
377	HWAAD63	1002	Production of VCAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to meaure the upregulation of cell surface VCAM-1 expression in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors, and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.
377	HWAAD63	1002	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or

			routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
HWABA81	1003	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al, FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
HWABA81	1003	Upregulation of CD152 and activation of T cells	CD152 FMAT. CD152 (a.k.a. CTLA4) expression is restricted to activated T cells. CD152 is a negative regulator of T cell proliferation. Reduced CD152 expression has been linked to hyperproliferative and autoimmune diseases. Overexpression of CD152 may lead to impaired immunoresponses. Assays for immunomodulatory proteins important in the maintenance of T cell homeostasis and expressed almost exclusively on CD4+ and CD8+ T cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate the activation of T cells, maintain T cell homeostasis, and/or mediate humoral or cell-mediated immunity. Exemplary assays that test for immunomodulatory proteins evaluate the upregulation of cell surface markers, such as CD152, and the activation of T cells. Such assays that may be used or routinely modified to test immunomodulatory activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include, for example, the assays disclosed in Miraglia et al., J Biomolecular Screening 4: 193-204 (1999); Rowland et al., "Lymphocytes: a practical approach" Chapter 6: 138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Chapter 6: 138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Chapter 6: 138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Chapter 6: 138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Chapter 6: 138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Chapter 6: 138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Chapter 6: 138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1999); Oostervegal et al., Chapter 6: 138-160 (2000); McCoy et al., Immunol Cell Biol 77(1):1-10 (1990); Oostervegal et al., Chapter 6: 138-160 (2000);
			the contents of each of which are herein incorporated by reference in its entirety. Human T cells that may be used according to these assays may be isolated using techniques disclosed herein or otherwise known in the art. Human T cells are primary human lymphocytes that mature in the thymus and express a T Cell receptor and CD3, CD4, or CD8. These cells mediate humoral or cell-

				mediated immunity and may be preactivated to enhance responsiveness to immunomodulatory factors.
379	HWADJ89	1004	Activation of transcription through serum response element in immune cells (such as T-cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate the serum response factors and modulate the expression of genes involved in growth. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension culture of T cells with cytotoxic activity.
379	HWADJ89	1004	Stimulation of insulin secretion from pancreatic beta cells.	Assays for measuring secretion of insulin are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate insulin secretion. For example, insulin secretion is measured by FMAT using anti-rat insulin antibodies. Insulin secretion from pancreatic beta cells is upregulated by glucose and also by certain proteins/peptides, and disregulation is a key component in diabetes. Exemplary assays that may be used or routinely modified to test for stimulation of insulin secretion (from pancreatic cells) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Ahren, B., et al., Am J Physiol, 277(4 Pt 2):R959-66 (1999); Li, M., et al., Endocrinology, 138(9):3735-40 (1997); Kim, K.H., et al., FEBS Lett, 377(2):237-9 (1995); and, Miraglia S et. al., Journal of Biomolecular Screening, 4:193-204 (1999), the contents of each of which is herein incorporated by reference in its entirety. Pancreatic cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary pancreatic cells that may be used according to these assays include rat INS-1 cells. INS-1 cells are a semi-adherent cell line established from cells isolated from an X-ray induced rat transplantable insulinoma. These cells retain characteristics typical of native pancreatic beta cells including glucose inducible insulin secretion. References: Asfari et al. Endocrinology 1992 130:167.
380	HWBAR88	1005	Production of IL-8 by immune cells	Assay that measures the production of the chemokine interleukin-8 (IL-8) from immune cells (such as the EOL-1 human eosinophil cell line) are well known in the art (for example, measurement of

			(such as the human EOL-1 eosinophil cells)	IL-8 production by FMAT) and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit. Eosinophils are a type of immune cell important in allergic responses; they are recruited to tissues and mediate the inflammtory response of late stage allergic reaction. IL.8 is a strong immunomodulator and may have a potential proinflammatory role in immunological diseases and disorders (such as allergy and asthma).	Š
380	HWBAR88	1005	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forer et al., Biol Chem 379(8-9):1101-1110 (1998), Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirery. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions, they are recruited to tissues and mediate the inflammatory response of late stage allergic reactions, they are recruited to tissues and mediate the inflammatory response of late stage allergic reactions, they are recruited to tissues and mediate the inflammatory response of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in dexamethasone-induced apoptosis and activation of -Jun NH2-terminal kinase and p38 mitogenactivated potein kinase in human eosinophils." Clin Exp Immunol; Oct;122(1):20-7 (2000); Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils." J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR, Peb 2;187(3):415-25 (1998); J Allergy Clin Immunol in subminate of prednisolone to inhibit are herein incorporated by reference in its e	
381	HWBCB89	1006	Activation of transcription through GATA-3 response element	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of	ب

			in immune cells (such as mast cells).	the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays
				Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol
				Cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays include the HMC.1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
381	HWBCB89	9001	Activation of transcription	Assays for the activation of transcription through the NFKB response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention
			through NFKB response element	(including antibodies and agonists or antagonists of the invention) to regulate NFKB transcription factors and modulate expression of immunomodulatory genes. Exemplary assays for transcription
			in immune cells (such as T-cells).	through the NFKB response element that may be used or rountinely modified to test NFKB-response element activity of polypeptides of the invention (including antibodies and agonists or
				antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthom et al., Proc Natl Acad Sci USA
				85:6342-6346 (1988); Black et al., Virus Gnes 15(2):105-117 (1997); and Fraser et al., 29(3):838-
				Exemplary human T cells, such as the MOLT4, that may be used according to these assays are
381	HWBCB89	1006	Production of	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely
			ICAM-1	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or
				routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al,
				FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000),
				the contents of each of which is herein incorporated by fererence in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be
				, , , , , , , , , , , , , , , , , , , ,

				routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
381	HWBCB89	1006	Activation of transcription through serum response element in immune cells (such as natural killer cells).	Assays for the activation of transcription through the Serum Response Element (SRE) are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate serum response factors and modulate the expression of genes involved in growth and upregulate the function of growth-related genes in many cell types. Exemplary assays for transcription through the SRE that may be used or routinely modified to test SRE activity of the polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Benson et al., J Immunol 153(9):3862-3873 (1994); and Black et al., Virus Genes 12(2):105-117 (1997), the content of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary T cells that may be used according to these assays include the NK-YT cell line, which is a human natural killer cell line with cytolytic and cytotoxic activity.
382	HWBCP79	1007	Activation of Adipocyte ERK Signaling Pathway	Kinase assay. Kinase assays, for example an Elk-1 kinase assay, for ERK signal transduction that regulate cell proliferation or differentiation are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and differentiation. Exemplary assays for ERK kinase activity that may be used or routinely modified to test ERK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Le Marchand-Brustel Y, Exp Clin Endocrinol Diabetes 107(2):126-132 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Mouse adipocyte cells that may be used according to these assays include 3T3-L1 cells. 3T3-L1 is an adherent mouse preadipocyte cell line that is a continuous substrain of 3T3 fibroblast cells developed through clonal isolation and undergo a pre-adipocyte to adipose-like conversion under appropriate differentiation conditions known in the art.
382	HWBCP79	1007	Production of IL- 10 and activation	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and

			of T-celle	aconiete or antaconiete of the invention) to etimulate or inhibit production of II - 10 and/or activation
				of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of
_				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
				modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in Pobinson DS et al "Th-2 cyricines in allernic disease" Br Med Bull: 56 (4): 055.
	-			968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology &
				Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
				reference in their entirety. Exemplary cells that may be used according to these assays include Th2:
	·			cells. IL.10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells
				are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation
				and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and
				asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions
				using peripheral blood lymphocytes isolated from cord blood.
383	HWBDP28	1008	Activation of	This reporter assay measures activation of the NFAT signaling pathway in HMC-1 human mast cell
			transcription	line. Activation of NFAT in mast cells has been linked to cytokine and chemokine production.
			through NFAT	Assays for the activation of transcription through the Nuclear Factor of Activated T cells (NFAT)
			response element	response element are well-known in the art and may be used or routinely modified to assess the
			in immune cells	ability of polypeptides of the invention (including antibodies and agonists or antagonists of the
			(such as mast	invention) to regulate NFAT transcription factors and modulate expression of genes involved in
			cells).	immunomodulatory functions. Exemplary assays for transcription through the NFAT response
				element that may be used or routinely modified to test NFAT-response element activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
				include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in
				Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); De
-				Boer et al., Int J Biochem Cell Biol 31(10):1221-1236 (1999); Ali et al., J Immunol 165(12):7215-
				7223 (2000); Hutchinson and McCloskey, J Biol Chem 270(27):16333-16338 (1995), and Turner et
				al., J Exp Med 188:527-537 (1998), the contents of each of which are herein incorporated by
				reference in its entirety. Mast cells that may be used according to these assays are publicly available
				(e.g., through the ATCC). Exemplary human mast cells that may be used according to these assays
				include the HMC-1 cell line, which is an immature human mast cell line established from the
				peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature
				mast cells.
383	HWBDP28	1008	Production of	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely
			ICAM-1	modified to assess the ability of polypeptides of the invention (including antibodies and agonists or

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				antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Takacs P, et al., FASEB J, 15(2):279-281 (2001); and, Miyamoto K, et al., Am J Pathol, 156(5):1733-1739 (2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that may be used according to these assays are publicly available (e.g., through the ATCC) and/or may be routinely generated. Exemplary cells that may be used according to these assays include microvascular endothelial cells (MVEC).
384	HWBFX31	1009	Regulation of transcription of Malic Enzyme in adipocytes	Assays for the regulation of transcription of Malic Enzyme are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate transcription of Malic Enzyme, a key enzyme in lipogenesis. Malic enzyme is involved in lipogenesisand its expression is stimulted by insulin. ME promoter contains two direct repeat (DR 1)- like elements MEp and MEd identified as putative PPAR response elements. ME promoter may also responds to AP1 and other transcription factors. Exemplary assays that may be used or routinely modified to test for regulation of transcription of Malic Enzyme (in adipoocytes) by polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in: Streeper, R.S. et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, 12(11):1778-91 (1998); Garcia-Jimenez, C., et al., Mol Endocrinol, al., J Biol Chem, 274(25):17997-8004 (1999); Ijpenberg, A., et al., J Biol Chem, 272(32):20108-20117 (1997); Berger, et al., Gene 66:1-10 (1988); and, Cullen, B., et al., Methods in Enzymol. 216:362-368 (1992), the contents of each of which is herein incorporated by reference in its entirety. Hepatocytes that may be used according to these assays includes the H4IIE rat liver hepatoma cell line.
385	HWHHL34	1010	Activation of transcription through cAMP response element in immune cells (such as T-cells).	Assays for the activation of transcription through the cAMP response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to increase cAMP and regulate CREB transcription factors, and modulate expression of genes involved in a wide variety of cell functions. Exemplary assays for transcription through the cAMP response element that may be used or routinely modified to test cAMP-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Black et al., Virus Genes 15(2):105-117 (1997); and Belkowski et al., Immunol 161(2):659-665 (1998), the contents of each of which

				and Leavis in a constant to an formancia its aminet. The all that may be seed in a to the
				assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is a suspension culture of IL-2 dependent cytotoxic T cells.
385	HWHHL34	1010	Activation of transcription through GATA-3 response element in immune cells (such as mast cells).	This reporter assay measures activation of the GATA-3 signaling pathway in HMC-1 human mast cell line. Activation of GATA-3 in mast cells has been linked to cytokine and chemokine production. Assays for the activation of transcription through the GATA3 response element are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate GATA3 transcription factors and modulate expression of mast cell genes important for immune response development. Exemplary assays for transcription through the GATA3 response element that may be used or routinely modified to test GATA3-response element activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include assays disclosed in Berger et al., Gene 66:1-10 (1998); Cullen and Malm, Methods in Enzymol 216:362-368 (1992); Henthorn et al., Proc Natl Acad Sci USA 85:6342-6346 (1988); Flavell et al., Cold Spring Harb Symp Quant Biol 64:563-571 (1999); Rodriguez-Palmero et al., Eur J Immunol 29(12):3914-3924 (1999); Zheng and Flavell, Cell 89(4):587-596 (1997); and Henderson et al., Mol cell Biol 14(6):4286-4294 (1994), the contents of each of which are herein incorporated by reference in its entirety. Mast cells that may be used according to these assays include the HMC-1 cell line, which is an immature human mast cell line established from the peripheral blood of a patient with mast cell leukemia, and exhibits many characteristics of immature mast cells.
386	нwнqsss	1011	Production of IL- 13 and activation of T-cells.	Assays for production of IL-13 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-13 and/or activation of T-cells. Exemplary assays for IL-13 production that may be used or routinely modified to test activity of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) include, for example, assays such as disclosed and/or cited in: Grunig, G, et al., "Requirement for IL-13 independently of IL-4 in Experimental asthma" Science;282: 2261-2263 (1998), and Wills-Karp M, et al., "Interleukin-13: central mediator of allergic asthma" Science; 282: 2258-2261 (1998); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL13, a Th2 type cytokine, is a potent stimulus for mucus production, airway hyper-responsiveness and

				allergic asthma. Th2 cells are a class of T cells that secrete IL4, IL.10, IL.13, IL.5 and IL.6. Factors that induce differentiation and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated in in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.
387	HWLIH65	1012	Activation of T-Cell p38 or JNK Signaling Pathway.	Kinase assay. JNK and p38 kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit immune cell (e.g. T-cell) proliferation, activation, and apoptosis. Exemplary assays for JNK and p38 kinase activity that may be used or routinely modified to test JNK and p38 kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. T cells that may be used according to these assays are publicly available (e.g., through the ATCC). Exemplary mouse T cells that may be used according to these assays include the CTLL cell line, which is an IL-2 dependent suspension-culture cell line with cytotoxic activity.
387	нwл.	1012	Production of VCAM in endothelial cells (such as human umbilical vein endothelial cells (HUVEC))	Assays for measuring expression of VCAM are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate VCAM expression. For example, FMAT may be used to meaure the upregulation of cell surface VCAM-1 expression in endothelial cells. Endothelial cells are cells that line blood vessels, and are involved in functions that include, but are not limited to, angiogenesis, vascular permeability, vascular tone, and immune cell extravasation. Exemplary endothelial cells that may be used according to these assays include human umbilical vein endothelial cells (HUVEC), which are available from commercial sources. The expression of VCAM (CD106), a membrane-associated protein, can be upregulated by cytokines or other factors, and contributes to the extravasation of lymphocytes, leucocytes and other immune cells from blood vessels; thus VCAM expression plays a role in promoting immune and inflammatory responses.
388	HYAAJ71	1013	Production of ICAM-1	Assays for measuring expression of ICAM-1 are well-known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to regulate ICAM-1 expression. Exemplary assays that may be used or routinely modified to measure ICAM-1 expression include assays disclosed in: Rolfe BE, et al., Atherosclerosis, 149(1):99-110 (2000); Panettieri RA Jr, et al., J Immunol, 154(5):2358-2365

				(1995); and, Grunstein MM, et al., Am J Physiol Lung Cell Mol Physiol, 278(6):L1154-L1163
				(2000), the contents of each of which is herein incorporated by reference in its entirety. Cells that
				may be used according to these assays are publicly available (e.g., through the ATCC) and/or may
				be routinely generated. Exemplary cells that may be used according to these assays include Aortic Smooth Muscle Cells (AOSMC); such as bovine AOSMC.
389	HYBAR01	1014	Production of	MCP-1 FMAT. Assays for immunomodulatory proteins that are produced by a large variety of cells
			MCP-1	and act to induce chemotaxis and activation of monocytes and T cells are well known in the art and
-				may be used or routinely modified to assess the ability of polypeptides of the invention (including
				antibodies and agonists or antagonists of the invention) to mediate immunomodulation, induce
				chemotaxis, and modulate immune cell activation. Exemplary assays that test for
				immunomodulatory proteins evaluate the production of cell surface markers, such as monocyte
	•			chemoattractant protein (MCP), and the activation of monocytes and T cells. Such assays that may
				be used or routinely modified to test immunomodulatory and diffferentiation activity of
				polypeptides of the invention (including antibodies and agonists or antagonists of the invention)
	-			include assays disclosed in Miraglia et al., J Biomolecular Screening 4:193-204(1999); Rowland et
				al., "Lymphocytes: a practical approach" Chapter 6:138-160 (2000); Satthaporn and Eremin, J R
				Coll Surg Ednb 45(1):9-19 (2001); and Verhasselt et al., J Immunol 158:2919-2925 (1997), the
				contents of each of which are herein incorporated by reference in its entirety. Human dendritic cells
				that may be used according to these assays may be isolated using techniques disclosed herein or
				otherwise known in the art. Human dendritic cells are antigen presenting cells in suspension
				culture, which, when activated by antigen and/or cytokines, initiate and upregulate T cell
				proliferation and functional activities.
389	HYBAR01	1014	Production of IL-	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used
			10 and activation	or routinely modified to assess the ability of polypeptides of the invention (including antibodies and
			of T-cells.	agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation
				of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of
				polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to
				modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed
				and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-
				968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology &
				Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by
				reference in their entirety. Exemplary cells that may be used according to these assays include Th2
	_			cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells
				are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation

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and activation of Th2 cells play a major role in the initiation and pathogenesis of allergy and asthma. Primary T helper 2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Assays for production of IL-10 and activation of T-cells are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to stimulate or inhibit production of IL-10 and/or activation of T-cells. Exemplary assays that may be used or routinely modified to assess the ability of polypeptides and antibodies of the invention (including agonists or antagonists of the invention) to modulate IL-10 production and/or T-cell proliferation include, for example, assays such as disclosed and/or cited in: Robinson, DS, et al., "Th-2 cytokines in allergic disease" Br Med Bull; 56 (4): 956-968 (2000), and Cohn, et al., "T-helper type 2 cell-directed therapy for asthma" Pharmacology & Therapeutics; 88: 187-196 (2000); the contents of each of which are herein incorporated by reference in their entirety. Exemplary cells that may be used according to these assays include Th2 cells. IL10 secreted from Th2 cells may be measured as a marker of Th2 cell activation. Th2 cells are a class of T cells that secrete IL4, IL10, IL13, IL5 and IL6. Factors that induce differentiation and activation of Th2 cells are generated via in vitro culture under Th2 polarizing conditions using peripheral blood lymphocytes isolated from cord blood.	Kinase assay. JNK kinase assays for signal transduction that regulate cell proliferation, activation, or apoptosis are well known in the art and may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to promote or inhibit cell proliferation, activation, and apoptosis. Exemplary assays for JNK kinase activity that may be used or routinely modified to test JNK kinase-induced activity of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) include the assays disclosed in Forrer et al., Biol Chem 379(8-9):1101-1110 (1998); Gupta et al., Exp Cell Res 247(2): 495-504 (1999); Kyriakis JM, Biochem Soc Symp 64:29-48 (1999); Chang and Karin, Nature 410(6824):37-40 (2001); and Cobb MH, Prog Biophys Mol Biol 71(3-4):479-500 (1999); the contents of each of which are herein incorporated by reference in its entirety. Exemplary cells that may be used according to these assays include eosinophils. Eosinophils are important in the late stage of allergic reactions; they are recruited to tissues and mediate the inflammatory response of late stage allergic reactions. Moreover, exemplary assays that may be used or routinely modified to assess the ability of polypeptides of the invention (including antibodies and agonists or antagonists of the invention) to modulate signal transduction, cell proliferation, activation, or apoptosis in eosinophils include assays disclosed and/or cited in: Zhang JP, et al., "Role of caspases in
	Production of IL- 10 and activation of T-cells.	Activation of JNK Signaling Pathway in immune cells (such as eosinophils).
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	dexamethasone-induced apoptosis and activation of c-Jun NH2-terminal kinase and p38 mitogen-
	activated protein kinase in human eosinophils" Clin Exp Immunol; Oct; 122(1):20-7 (2000);
	Hebestreit H, et al., "Disruption of fas receptor signaling by nitric oxide in eosinophils" J Exp Med;
 -	Feb 2,187(3):415-25 (1998); J Allergy Clin Immunol 1999 Sep;104(3 Pt 1):565-74; and, Sousa AR,
	et al., "In vivo resistance to corticosteroids in bronchial asthma is associated with enhanced
	phosyphorylation of JUN N-terminal kinase and failure of prednisolone to inhibit JUN N-terminal
	kinase phosphorylation" J Allergy Clin Immunol; Sep;104(3 Pt 1):565-74 (1999); the contents of
	each of which are herein incorporated by reference in its entirety.

Table 1E: Polynucleotides encoding polypeptides of the present invention can be used in assays to test for one or more biological activities. One such biological activity which may be tested includes the ability of polynucleotides and polypeptides of the invention to stimulate up-regulation or down-regulation of expression of particular genes and proteins. Hence, if polynucleotides and polypeptides of the present invention exhibit activity in altering particular gene and protein expression patterns, it is likely that these polynucleotides and polypeptides of the present invention may be involved in, or capable of effecting changes in, diseases associated with the altered gene and protein expression profiles. Hence, polynucleotides, polypeptides, or antibodies of the present invention could be used to treat said associated diseases.

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TaqMan® assays may be performed to assess the ability of polynucleotides (and polypeptides they encode) to alter the expression pattern of particular "target" genes. TaqMan® reactions are performed to evaluate the ability of a test agent to induce or repress expression of specific genes in different cell types. TaqMan® gene expression quantification assays ("TaqMan® assays") are well known to, and routinely performed by, those of ordinary skill in the art. TaqMan® assays are performed in a two step reverse transcription / polymerase chain reaction (RT-PCR). In the first (RT) step, cDNA is reverse transcribed from total RNA samples using random hexamer primers. In the second (PCR) step, PCR products are synthesized from the cDNA using gene specific primers.

To quantify gene expression the Taqman® PCR reaction exploits the 5' nuclease activity of AmpliTaq Gold® DNA Polymerase to cleave a Taqman® probe (distinct from the primers) during PCR. The Taqman® probe contains a reporter dye at the 5'-end of the probe and a quencher dye at the 3' end of the probe. When the probe is intact, the proximity of the reporter dye to the quencher dye results in suppression of the reporter fluorescence. During PCR, if the target of interest is present, the probe specifically anneals between the forward and reverse primer sites. AmpliTaq Fold DNA Polymerase then cleaves the probe between the reporter and quencher when the probe hybridizes to the target, resulting in increased fluorescence of the reporter (see Figure 2). Accumulation of PCR products is detected directly by monitoring the increase in fluorescence of the reporter dye.

After the probe fragments are displaced from the target, polymerization of the strand continues. The 3'-end of the probe is blocked to prevent extension of the probe during PCR. This process occurs in every cycle and does not interfere with the exponential accumulation of product. The increase in fluorescence signal is detected only if the target sequence is complementary to the probe and is amplified during PCR. Because of these requirements, any nonspecific amplification is not detected.

For test sample preparation, vector controls or constructs containing the coding sequence for the gene of interest are transfected into cells, such as for example 293T cells, and supernatants collected after 48 hours. For cell treatment and RNA isolation, multiple primary human cells or

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human cell lines are used; such cells may include but are not limited to, Normal Human Dermal Fibroblasts, Aortic Smooth Muscle, Human Umbilical Vein Endothelial Cells, HepG2, Daudi, Jurkat, U937, Caco, and THP-1 cell lines. Cells are plated in growth media and growth is arrested by culturing without media change for 3 days, or by switching cells to low serum media and incubating overnight. Cells are treated for 1, 6, or 24 hours with either vector control supernatant or sample supernatant (or purified/partially purified protein preparations in buffer). Total RNA is isolated; for example, by using Trizol extraction or by using the Ambion RNAqueous(TM)-4PCR RNA isolation system. Expression levels of multiple genes are analyzed using TAQMAN, and expression in the test sample is compared to control vector samples to identify genes induced or repressed. Each of the above described techniques are well known to, and routinely performed by, those of ordinary skill in the art.

Table 1E indicates particular disease classes and preferred indications for which polynucleotides, polypeptides, or antibodies of the present invention may be used in detecting, diagnosing, preventing, treating and/or ameliorating said diseases and disorders based on "target" gene expression patterns which may be up- or down-regulated by polynucleotides (and the encoded polypeptides) corresponding to each indicated cDNA Clone ID (shown in Table 1E, Column 2).

Thus, in preferred embodiments, the present invention encompasses a method of detecting, diagnosing, preventing, treating, and/or ameliorating a disease or disorder listed in the "Disease Class" and/or "Preferred Indication" columns of Table 1E; comprising administering to a patient in which such detection, diagnosis, prevention, or treatment is desired a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof) in an amount effective to detect, diagnose, prevent, treat, or ameliorate the disease or disorder. The first and second columns of Table 1D show the "Gene No." and "cDNA Clone ID No.", respectively, indicating certain nucleic acids and proteins (or antibodies against the same) of the invention (including polynucleotide, polypeptide, and antibody fragments or variants thereof) that may be used in detecting, diagnosing, preventing, treating, or ameliorating the disease(s) or disorder(s) indicated in the corresponding row in the "Disease Class" or "Preferred Indication" Columns of Table 1E.

In another embodiment, the present invention also encompasses methods of detecting, diagnosing, preventing, treating, or ameliorating a disease or disorder listed in the "Disease Class" or "Preferred Indication" Columns of Table 1E; comprising administering to a patient combinations of the proteins, nucleic acids, or antibodies of the invention (or fragments or variants thereof), sharing similar indications as shown in the corresponding rows in the "Disease Class" or "Preferred Indication" Columns of Table 1E.

The "Disease Class" Column of Table 1E provides a categorized descriptive heading for diseases, disorders, and/or conditions (more fully described below) that may be detected, diagnosed,

5 prevented, treated, or ameliorated by a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof).

The "Preferred Indication" Column of Table 1E describes diseases, disorders, and/or conditions that may be detected, diagnosed, prevented, treated, or ameliorated by a protein, nucleic acid, or antibody of the invention (or fragment or variant thereof).

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The "Cell Line" and "Exemplary Targets" Columns of Table 1E indicate particular cell lines and target genes, respectively, which may show altered gene expression patterns (i.e., up- or down-regulation of the indicated target gene) in Taqman assays, performed as described above, utilizing polynucleotides of the cDNA Clone ID shown in the corresponding row. Alteration of expression patterns of the indicated "Exemplary Target" genes is correlated with a particular "Disease Class" and/or "Preferred Indication" as shown in the corresponding row under the respective column headings.

The "Exemplary Accessions" Column indicates GenBank Accessions (available online through the National Center for Biotechnology Information (NCBI) at http://www.ncbi.nlm.nih.gov/) which correspond to the "Exemplary Targets" shown in the adjacent row.

The recitation of "Cancer" in the "Disease Class" Column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof) may be used for example, to detect, diagnose, prevent, treat, and/or ameliorate neoplastic diseases and/or disorders (e.g., leukemias, cancers, etc., as described below under "Hyperproliferative Disorders").

The recitation of "Immune" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof), may be used for example, to detect, diagnose, prevent, treat, and/or ameliorate diseases and/or disorders relating to neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), blood disorders (e.g., as described below under "Immune Activity" "Cardiovascular Disorders" and/or "Blood-Related Disorders"), and infections (e.g., as described below under "Infectious Disease").

The recitation of "Angiogenesis" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof), may be used for example, to detect, diagnose, treat, prevent, and/or ameliorate diseases and/or disorders relating to neoplastic diseases (e.g., as described below under "Hyperproliferative Disorders"), diseases and/or disorders of the cardiovascular system (e.g., as described below under "Cardiovascular Disorders"), diseases and/or disorders involving cellular and genetic abnormalities (e.g., as described below under "Diseases at the Cellular Level"), diseases and/or disorders involving angiogenesis (e.g., as described below under "Anti-Angiogenesis Activity"), to promote or inhibit cell or tissue regeneration (e.g., as described below under

5 "Regeneration"), or to promote wound healing (e.g., as described below under "Wound Healing and Epithelial Cell Proliferation").

The recitation of "Diabetes" in the "Disease Class" column indicates that the corresponding nucleic acid and protein, or antibody against the same, of the invention (or fragment or variant thereof), may be used for example, to detect, diagnose, treat, prevent, and/or ameliorate diabetes (including diabetes mellitus types I and II), as well as diseases and/or disorders associated with, or consequential to, diabetes (e.g. as described below under "Endocrine Disorders," "Renal Disorders," and "Gastrointestinal Disorders").

TABLE 1E

Gene	cDNA Clone		Preferred Indications	Cell Line	Exemplary	Exemplary
No.	Ð	Class			Targets	Accessions
126	н н н н н н н н н н н н н н н н н н н	Immune	Highly preferred indications include immunological disorders such as described herein under the heading "Immune Activity" and/or	TF-1	CD40	gb AJ300189 H
	-		"Blood-Related Disorders" (particularly including, but not limited			gb Z22576 HS
	-		to, immune disorders involving erythrocytes). Highly preferred			CD69GNA
			embodiments of the invention include methods of preventing,			
			detecting, diagnosing, treating and/or ameliorating disorders of the			-
			immune system (particularly including, but not limited to, immune			
	-		disorders involving erythrocytes). (The TF-1 cell line is a human			
			erythroblast cell line available through the ATCC as cell line			
100	OSCOCCITIES OF THE PROPERTY OF	1	number Cal-2003),		34,01	TOTALOGO AND I
07.1		Immune	Highly preferred indications include immunological disorders such	093/	ICAM	ISH 06690X q8
			as described herein under the heading "Immune Activity" and/or		IRF1	CAMI
			"Blood-Related Disorders" (particularly including, but not limited		LTBR	gb X14454 HSI
			to, immune disorders involving monocytes). Highly preferred			RFI
			embodiments of the invention include methods of preventing,			gb[AK027080]
			detecting, diagnosing, treating and/or ameliorating disorders of the			AK027080
			immune system (particularly including, but not limited to, immune			
			disorders involving monocytes). (The U937 cell line is a human			
			monocyte cell line available through the ATCC as cell line number			
			CRL-1593.2).			
171	HKACD58	Immune	Highly preferred indications include immunological disorders such	AOSMC	VCAM	gb A30922 A30
			as described herein under the heading "Immune Activity" and/or			922
			"Blood-Related Disorders" (particularly including, but not limited			
			to, immune disorders involving muscle tissues and the			
			cardiovascular system (e.g. heart, lungs, circulatory system)).			
			Highly preferred embodiments of the invention include methods of			
			preventing, detecting, diagnosing, treating and/or ameliorating			
			disorders of the immune system (particularly including, but not			

			limited to, immune disorders involving muscle tissue or the cardiovascular system). (AOSMC cells are human aortic smooth muscle cells).			
171	HKACD58	Immune	red indications include immunological disorders such herein under the heading "Immune Activity" and/or ed Disorders" (particularly including, but not limited isorders involving the B-cells). Highly preferred of the invention include methods of preventing, gnosing, treating and/or ameliorating disorders of the em (particularly including, but not limited to, immune olving B-cells). (The Daudi cell line is a human B cell line available through the ATCC as cell line -213).	Daudi	CD40	gb AJ300189 H SA30018
171	HKACD58	Immune		HUVEC	ICAM Rag l	gb X06990 HSI CAM1 gb M29474 HU MRAG1
171	HKACD58	Гттипе	r ch	Liver	CD28	gb AF222342 AF222342
171	HKACD58	Immune	preferred indications include immunological disorders such ibed herein under the heading "Immune Activity" and/or Related Disorders" (particularly including, but not limited	NHDF	CXCR3 GATA1 116	gb Z79783 HS CKRL2 gb X17254 HS

			to, immune disorders involving the skin). Highly preferred embodiments of the invention include methods of preventing, detecting, diagnosing, treating and/or ameliorating disorders of the immune system (particularly including, but not limited to, immune disorders involving the skin). (NHDF cells are normal human dermal fibroblasts).		VCAM	ERYF1 gb X04403 HS 26KDAR gb A30922 A30 922
171	HKACD58	Immune	Highly preferred indications include immunological disorders such as described herein under the heading "Immune Activity" and/or "Blood-Related Disorders" (particularly including, but not limited to, immune disorders involving monocytes). Highly preferred embodiments of the invention include methods of preventing, detecting, diagnosing, treating and/or ameliorating disorders of the immune system (particularly including, but not limited to, immune disorders involving monocytes). (The THPI cell line is a human monocyte cell line available through the ATCC as cell line number TIB-202).	PI	CIS3	gb AB006967 AB006967
171	HKACD58	Immune	Highly preferred indications include immunological disorders such as described herein under the heading "Immune Activity" and/or "Blood-Related Disorders" (particularly including, but not limited to, immune disorders involving monocytes). Highly preferred embodiments of the invention include methods of preventing, detecting, diagnosing, treating and/or ameliorating disorders of the immune system (particularly including, but not limited to, immune disorders involving monocytes). (The U937 cell line is a human monocyte cell line available through the ATCC as cell line number CRL-1593.2).	37	CD69 TNF	gb Z22576 HS CD69GNA gb AJ270944 H SA27094
312	HSDSB09	Immune	Highly preferred indications include immunological disorders such as described herein under the heading "Immune Activity" and/or "Blood-Related Disorders" (particularly including, but not limited to, immune disorders involving muscle tissues and the cardiovascular system (e.g. heart, lungs, circulatory system)). Highly preferred embodiments of the invention include methods of preventing, detecting, diagnosing, treating and/or ameliorating disorders of the immune system (particularly including, but not	SMC	CCR3 CCR4 CD25 CD30 CD40 CTLA4 IL5 Rag1	gb AB023887 AB023887 gb AB023888 AB023888 gb X03137 HSI L2RG7

			limited to, immune disorders involving muscle tissue or the cardiovascular system). (AOSMC cells are human aortic smooth muscle cells).		VCAM	SA30018 gb AF316875 AF316875
						gb X12705 HS BCDFIA
				•	•	gb M29474 HU MRAG1
			·	·		gb A30922 A30 922
312	HSDSB09	Immune	용	Caco-2	c-maf	gb AF055377
			as described herein under the heading "Immune Activity" and/or "Blood-Related Disorders" (particularly including, but not limited		GATA3 ICAM	AF055377 gb X55037 HS
			to, immune disorders involving the cells of the gastrointestinal	•	Rag1	GATA3
			tract). Highly preferred embodiments of the invention include	-		ISH 06690X q8
			methods of preventing, detecting, diagnosing, treating and/or			CAMI
			ameliorating disorders of the immune system (particularly			gb[M29474 HU
			including, but not limited to, immune disorders involving cells of			MKAGI
			the gastrointestinal tract). (The Caco-2 cell line is a human			
			colorectal adenocarcinoma cell line available infougn the ATCC as cell line number HTB-37).			•
312	HSDSB09	Immune	nclude immunological disorders such	Daudi	TNF	gb AJ270944 H
			as described herein under the heading "Immune Activity" and/or			SA27094
			"Blood-Related Disorders" (particularly including, but not limited			
			to, immune disorders involving the B-cells). Highly preferred			
			embodiments of the invention include methods of preventing,			
			detecting, diagnosing, treating and/or ameliorating disorders of the			
			immune system (particularly including, but not limited to, immune			
			disorders involving B-cells). (The Daudi cell line is a human B			
			lymphoblast cell line available through the ATCC as cell line			
	OCCUPACIT		number CCL-213).	,	COLO	1110000000
312	HSDSB09	Immune	Highly preferred indications include immunological disorders such H9		CIS3	gb AB006967
			as described nerein under the neading immune Activity and/or		Kagi	AB00090/
			"Blood-Related Disorders" (particularly including, but not limited			gb[M29474JHU
			to, infinitine disorders involving the 1-cells). Enginy preferred			MKAGI

		embodiments of the invention include methods of preventing, detecting, diagnosing, treating and/or ameliorating disorders of the immune system (particularly including, but not limited to, immune disorders involving T-cells). (The H9 cell line is a human T lymphocyte cell line available through the ATCC as cell line			
HSDSB09	Immune	s such I/or nited ude	HEK293	CCR3 CCR4 CD25 CD30 CD40	gb AB023887 AB023887 gb AB023888 AB023888 gb X03137 HSI
		methods of preventing, detecting, diagnosing, treating and/or ameliorating disorders of the irmmune system (particularly including, but not limited to, immune disorders involving epithelial cells or the renal system). (The 293 cell line is a human embryonal kidney epithelial cell line available through the ATCC as cell line number CRL-1573).		CILA4 GATA3 Rag1 TNF VCAM	EZKG/ gb AJ300189 H SA30018 gb AF316875 AF316875 eb X55037 HS
					GATA3 gb M29474 HU MRAG1 gb AJ270944 H SA27094 gb A30922 A30 922
HSDSB09	Immune	Highly preferred indications include immunological disorders such as described herein under the heading "Immune Activity" and/or "Blood-Related Disorders" (particularly including, but not limited to, immune disorders involving endothelial cells). Highly preferred embodiments of the invention include methods of preventing, detecting, diagnosing, treating and/or ameliorating disorders of the immune system (particularly including, but not limited to, immune disorders involving endothelial cells).	HUVEC	CD40 ICAM IL.10 Rag.1 Rag.2 TNF	gb AJ300189 H SA30018 gb X06990 HSI CAM1 gb AF055467 AF055467 gb M29474 HU MRAG1

Immune Highly preferred indications include immunological disorders such as described herein under the heading "Immune Activity" and/or "Blood-Related Disorders" (particularly including, but not limited to, immune disorders involving T-cells). Highly preferred
embodim
Annual system (particularly including, but not innuced to, infinunce disorders involving T-cells). (The Jurkat cell line is a human T lymphocyte cell line available through the ATCC as cell line number TB-152).
Immune Highly pre as describe
to, immune Highly pred
disorders of limited to, i system).
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"Blood-Kel to, immune embodimen
immune sys disorders in
cell line av 1582).
Immune Highly pre

	·		"Blood-Related Disorders" (particularly including, but not limited to, immune disorders involving the skin). Highly preferred embodiments of the invention include methods of preventing, detecting, diagnosing, treating and/or ameliorating disorders of the immune system (particularly including, but not limited to, immune disorders involving the skin). (NHDF cells are normal human dermal fibroblasts)	116		gb AJ300189 H SA30018 gb X04403 HS 26KDAR
312	HSDSB09	Immune	Highly preferred indications include immunological disorders such as described herein under the heading "Immune Activity" and/or MC "Blood-Related Disorders" (particularly including, but not limited to, immune disorders involving the central nervous system). Highly preferred embodiments of the invention include methods of preventing, detecting, diagnosing, treating and/or ameliorating disorders of the immune system (particularly including, but not limited to, immune disorders involving the central nervous sytem). (The SK-N-MC neuroblastoma cell line is a cell line derived from human brain tissue and is available through the ATCC as cell line number HTB-10).	N- c-maf CIS3 oblas TNF	44	gb AF055377 AF055377 gb AB006967 AB006967 gb AJ270944 H SA27094
312	HSDSB09	Іттипе	Highly preferred indications include immunological disorders such as described herein under the heading "Immune Activity" and/or "Blood-Related Disorders" (particularly including, but not limited to, immune disorders involving T-cells). Highly preferred embodiments of the invention include methods of preventing, detecting, diagnosing, treating and/or ameliorating disorders of the immune system (particularly including, but not limited to, immune disorders involving T-cells). (The SUPT cell line is a human T-cell line).	T TNF VCAM	M	gb AJ270944 H SA27094 gb A30922 A30 922
312	HSDSB09	Immune	Highly preferred indications include immunological disorders such as described herein under the heading "Immune Activity" and/or "Blood-Related Disorders" (particularly including, but not limited to, immune disorders involving monocytes). Highly preferred embodiments of the invention include methods of preventing, detecting, diagnosing, treating and/or ameliorating disorders of the immune system (particularly including, but not limited to, immune	CCR3 CD40 GATA3 ICAM IL5 Rag2 VCAM	.3 0 7.43 M	gb AB023887 AB023887 gb AJ300189 H SA30018 gb X55037 HS GATA3 gb X06990 HSI

			disorders involving monocytes). (The THP1 cell line is a human monocyte cell line available through the ATCC as cell line number TB-202).		CAM1 gb X12705 HS BCDFIA
					gb AY011962 AY011962
					gb A30922 A30 922
312	HSDSB09	Immune	Highly preferred indications include immunological disorders such U937 as described herein under the heading "Immune Activity" and/or	IL1B	gb X02532 HSI L1BR
			"Blood-Related Disorders" (particularly including, but not limited		
			to, infiniting disorders involving monocytes). August preferred embodiments of the invention include methods of preventing.		
			detecting, diagnosing, treating and/or ameliorating disorders of the		
			immune system (particularly including, but not limited to, immune		
			disorders involving monocytes). (The U937 cell line is a human		
			monocyte cell line available through the ATCC as cell line number		
			CRL-1593.2).		
373	HUKBT29	Immune	Highly preferred indications include immunological disorders such U937	69CO	gb Z22576 HS
			as described herein under the heading "Immune Activity" and/or		CD69GNA
			"Blood-Related Disorders" (particularly including, but not limited		
			to, immune disorders involving monocytes). Highly preferred		
			embodiments of the invention include methods of preventing,		
			detecting, diagnosing, treating and/or ameliorating disorders of the		
			immune system (particularly including, but not limited to, immune		
			disorders involving monocytes). (The U937 cell line is a human		
_			monocyte cell line available through the ATCC as cell line number		•
			CRL-1593.2).		

Table 2 further characterizes certain encoded polypeptides of the invention, by providing the results of comparisons to protein and protein family databases. The first column provides a unique clone identifier, "Clone ID NO:", corresponding to a cDNA clone disclosed in Table 1A and/or Table 1B. The second column provides the unique contig identifier, "Contig ID:" which allows correlation with the information in Table 1B. The third column provides the sequence identifier, "SEQ ID NO:", for the contig polynucleotide sequences. The fourth column provides the analysis method by which the homology/identity disclosed in the Table was determined. The fifth column provides a description of the PFAM/NR hit identified by each analysis. Column six provides the accession number of the PFAM/NR hit disclosed in the fifth column. Column seven, score/percent identity, provides a quality score or the percent identity, of the hit disclosed in column five. Comparisons were made between polypeptides encoded by polynucleotides of the invention and a non-redundant protein database (herein referred to as "NR"), or a database of protein families (herein referred to as "PFAM"), as described below.

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The NR database, which comprises the NBRF PIR database, the NCBI GenPept database, and the SIB SwissProt and TrEMBL databases, was made non-redundant using the computer program nrdb2 (Warren Gish, Washington University in Saint Louis). Each of the polynucleotides shown in Table 1B, column 3 (e.g., SEQ ID NO:X or the 'Query' sequence) was used to search against the NR database. The computer program BLASTX was used to compare a 6-frame translation of the Query sequence to the NR database (for information about the BLASTX algorithm please see Altshul et al., J. Mol. Biol. 215:403-410 (1990), and Gish and States, Nat. Genet. 3:266-272 (1993). A description of the sequence that is most similar to the Query sequence (the highest scoring 'Subject') is shown in column five of Table 2 and the database accession number for that sequence is provided in column six. The highest scoring 'Subject' is reported in Table 2 if (a) the estimated probability that the match occurred by chance alone is less than 1.0e-07, and (b) the match was not to a known repetitive element. BLASTX returns alignments of short polypeptide segments of the Query and Subject sequences which share a high degree of similarity; these segments are known as High-Scoring Segment Pairs or HSPs. Table 2 reports the degree of similarity between the Query and the Subject for each HSP as a percent identity in Column 7. The percent identity is determined by dividing the number of exact matches between the two aligned sequences in the HSP, dividing by the number of Query amino acids in the HSP and multiplying by 100. The polynucleotides of SEQ ID NO:X which encode the polypeptide sequence that generates an HSP are delineated by columns 8 and 9 of Table 2.

The PFAM database, PFAM version 2.1, (Sonnhammer, Nucl. Acids Res., 26:320-322, 1998))consists of a series of multiple sequence alignments; one alignment for each protein family. Each multiple sequence alignment is converted into a probability model called a Hidden Markov Model, or HMM, that represents the position-specific variation among the sequences that make up

the multiple sequence alignment (see, e.g., Durbin, et al., Biological sequence analysis: probabilistic models of proteins and nucleic acids, Cambridge University Press, 1998 for the theory of HMMs). The program HMMER version 1.8 (Sean Eddy, Washington University in Saint Louis) was used to compare the predicted protein sequence for each Query sequence (SEQ ID NO:Y in Table 1B.1) to each of the HMMs derived from PFAM version 2.1. A HMM derived from PFAM version 2.1 was said to be a significant match to a polypeptide of the invention if the score returned by HMMER 1.8 was greater than 0.8 times the HMMER 1.8 score obtained with the most distantly related known member of that protein family. The description of the PFAM family which shares a significant match with a polypeptide of the invention is listed in column 5 of Table 2, and the database accession number of the PFAM hit is provided in column 6. Column 7 provides the score returned by HMMER version 1.8 for the alignment. Columns 8 and 9 delineate the polynucleotides of SEQ ID NO:X which encode the polypeptide sequence which show a significant match to a PFAM protein family.

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As mentioned, columns 8 and 9 in Table 2, "NT From" and "NT To", delineate the polynucleotides of "SEQ ID NO:X" that encode a polypeptide having a significant match to the PFAM/NR database as disclosed in the fifth column. In one embodiment, the invention provides a protein comprising, or alternatively consisting of, a polypeptide encoded by the polynucleotides of SEQ ID NO:X delineated in columns 8 and 9 of Table 2. Also provided are polynucleotides encoding such proteins, and the complementary strand thereto.

The nucleotide sequence SEQ ID NO:X and the translated SEQ ID NO:Y are sufficiently accurate and otherwise suitable for a variety of uses well known in the art and described further below. For instance, the nucleotide sequences of SEQ ID NO:X are useful for designing nucleic acid hybridization probes that will detect nucleic acid sequences contained in SEQ ID NO:X or the cDNA contained in ATCC Deposit No:Z. These probes will also hybridize to nucleic acid molecules in biological samples, thereby enabling immediate applications in chromosome mapping, linkage analysis, tissue identification and/or typing, and a variety of forensic and diagnostic methods of the invention. Similarly, polypeptides identified from SEQ ID NO:Y may be used to generate antibodies which bind specifically to these polypeptides, or fragments thereof, and/or to the polypeptides encoded by the cDNA clones identified in, for example, Table 1A and/or 1B.

Nevertheless, DNA sequences generated by sequencing reactions can contain sequencing errors. The errors exist as misidentified nucleotides, or as insertions or deletions of nucleotides in the generated DNA sequence. The erroneously inserted or deleted nucleotides cause frame shifts in the reading frames of the predicted amino acid sequence. In these cases, the predicted amino acid sequence diverges from the actual amino acid sequence, even though the generated DNA

sequence may be greater than 99.9% identical to the actual DNA sequence (for example, one base insertion or deletion in an open reading frame of over 1000 bases).

Accordingly, for those applications requiring precision in the nucleotide sequence or the amino acid sequence, the present invention provides not only the generated nucleotide sequence identified as SEQ ID NO:X, and a predicted translated amino acid sequence identified as SEQ ID NO:Y, but also a sample of plasmid DNA containing cDNA ATCC Deposit No:Z (e.g., as set forth in columns 2 and 3 of Table 1A and/or as set forth, for example, in Table 1B, 6, and 7). The nucleotide sequence of each deposited clone can readily be determined by sequencing the deposited clone in accordance with known methods. Further, techniques known in the art can be used to verify the nucleotide sequences of SEQ ID NO:X. The predicted amino acid sequence can then be verified from such deposits. Moreover, the amino acid sequence of the protein encoded by a particular clone can also be directly determined by peptide sequencing or by expressing the protein in a suitable host cell containing the deposited human cDNA, collecting the protein, and determining its sequence.

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	NT	То	777	396	1206	202	1352	1132	379	357 1368	1003	1114	329	619	329 786	234	518	1001	433	221	402	844
	ZN	From	10	202	1123	116	1200	413	188	211 1306	470	317	144	311	135	16	228	414	2	183	229	338
	Score/	Percent Identity	%001	46%	%09	100%	100%	%86 88%	100%	100% 95%	142.7	%001	35.6	100%	93%	100%	%96	% 66	93%	100%	72%	100%
	PFam/NR	Accession Number	O9NYD1	LAXN6D					бэлнеэ	pir JE0383 JE038	PF00822	AAH19290	PF00822	gb AAC51364.1		COP2_HUMAN		Q9UKG4		Q9BT67		
		PFam/NR Description	(Q9NYDI) G-PROTEIN-COUPLED RECEPTOR 48.	(Q9NXY7) CHONDROITIN 4-O-	SULFOTRANSFERASE (CHONDROITIN 4-0-	SULFOTRANS			(Q9UHE9) ZSIG9 PROTEIN (TRANSMEMBRANE PROTEIN 4).	NADH dehydrogenase (ubiquinone) (EC 1.6.5.3) chain NDUFB4 - human	PFAM: PMP-22/EMP/MP20/Claudin family	(AAH19290) Hypothetical 27.7 kDa protein (Fragment)	PFAM: PMP-22/EMP/MP20/Claudin family	(AF000959) transmembrane protein [Homo sapiens]		(015432) PROBABLE LOW-AFFINITY COPPER	UPTAKE PROTEIN 2 (HCT	(Q9UKG4) NA+/SULFATE COTRANSPORTER SUT-1.		(Q9BT67) UNKNOWN (PROTEIN FOR MGC:10924).		
	Analysis	Method	WUblastx. 64	WUblastx.	64				WUblastx.	WUblastx. 64	HMMER 2.1.1	WUblastx.	HMMER 2.1.1	blastx.2		WUblastx.	64	WUblastx.	64	WUblastx.	64	
	SEQ ID	ÿ×	11	403					404	17	18		405			19		20		23		
	Contig	ë	884134	589947					637786	637482	891114		731877			381942		561996		778820		
TABLE 2	cDNA	Clone ID	H2CBU83	H6EAB28					H6EDX46	HACBD91	HACCI17		HACCI17			HAGAI85		HAGAQ26		HAGFY16		

														1100	72/002	
LN	To	720	1261	1465	617	665	48	1869	999	686 573	1956	2757	2756	823	418	1017
NT	From	1	290	83	294	120 557	219	25	1	576 4	1807	1495	1503	59	59	70
Score/	Percent Identity	<i>%</i> 98	%66	100%	126.6	91% 93%	%69	%66	%08	94% 81%	22.9	%86	93%	100%	%66	83%
PFam/NR	Accession Number	gb AAG44248.1	629960	Q9H173	PF01039	ОЭНССО	SDIIGO	pir T08708 T087 08	9වපුල්ර	<u>රෟ</u> BGQ6	PF00781	Q9NP48	sp Q9NP48 Q9N P48	AAH07438	gb AAD26810.1 AF119297_1	AAH08720
	PFam/NR Description	(AF220209) Nedd4 WW domain-binding protein 5 [Mus musculus]	(Q96G79) Similar to RIKEN cDNA 2610030116 gene.	(Q9H173) SIL1 PROTEIN PRECURSOR.	PFAM: Carboxyl transferase domain	(Q9HCC0) NON-BIOTIN CONTAINING SUBUNIT OF 3-METHYLCROTONYL-COA CARBOX	(Q91IG5) UBIQUITIN SPECIFIC PROTEASE (FRAGMENT).	hypothetical protein DKFZp564D116.1 - human (fragment)	(Q9BGQ6) HYPOTHETICAL 30.3 KDA PROTEIN.	(Q9BGQ6) HYPOTHETICAL 30.3 KDA PROTEIN.	PFAM: Diacylglycerol kinase catalytic domain (presumed)	(Q9NP48) PUTATIVE LIPID KINASE (CDNA FLJ10842 FIS, CLONE NT2RP4001343	PUTATIVE LIPID KINASE (CDNA FLJ10842 FIS, CLONE NT2RP4001343).	(AAH07438) Similar to RIKEN cDNA 2610511E22 gene.	(AF119297) neuroendocrine-specific protein-like protein 1 [Homo sapiens]	(AAH08720) Unknown (protein for MGC:8447).
Analysis	Method	blastx.2	WUblastx.	WUblastx.	HMMER 2.1.1	WUblastx. 64	WUblastx.	WUblastx. 64	WUblastx.	WUblastx.	HMMER 2.1.1	WUblastx. 64	blastx.2	WUblastx.	blastx.2	WUblastx.
SEQ ID	No. ×	407	25	56	409		28	29	30	410	31		412	33	414	415
Contig	ë	381964	727543	422672	872551		638516	618530	904749	985006	\$69506		823350	892971	852533	844216
cDNA	Clone ID	HAGFY16	HAIBP89	HAICP19	HAJAN23		HAJBR69	HAJBZ75	HAMFC93	HAMFC93	HAMFE15		HAMFE15	HAMGR28	HANGG89	HANGG89

W U U3	70500	-												_						C 170	1502/0	0211
Į,	To	1068	1310	817	850	263	1056	844	804	669	840	1280	593	999	610	1216	399	84	557	723	950	779
IN	From	490	27 70	251	59	54	982	266	109	99	811	1212	99	516	750	143	160	25	489	406	672	3
Score/	Percent Identity	51%	99% 40%	%66	91%	100%	36%	100%	% 66	100%	100%	39%	%86	%99	%89 %0L	93%	88%	%06	100%	100%	%9 6	100%
PFam/NR	Accession Number		AAH08720	Q9BZM1	AAH20263	AAH20263			1МЛЯ6О	AAH19903			AAH19903		О9Н728	095803	BAB27250			gb AAG43119.1 AF059620_1	Q9GZR7	890Н6О
	PFam/NR Description		(AAH08720) Unknown (protein for MGC:8447).	(Q9BZM1) GROUP XII SECRETED PHOSPHOLIPASE A2.	(AAH20263) Hypothetical 28.7 kDa protein.	(AAH20263) Hypothetical 28.7 kDa protein.			(Q9BUM1) UNKNOWN (PROTEIN FOR IMAGE:3050476) (FRAGMENT).	(AAH19903) Hypothetical 29.4 kDa protein (Fragment)			(AAH19903) Hypothetical 29.4 kDa protein (Fragment)		(Q9H728) CDNA: FLJ21463 FIS, CLONE COL04765.	(095803) HEPARAN SULFATE N-DEACETYLASE/N-SULFOTRANSFERASE 3.	+			(AF059620) My006 protein [Homo sapiens]	(Q9GZR7) HYPOTHETICAL 96.3 KDA PROTEIN (ATP-DEPENDENT RNA HELICASE) ((Q9H068) HYPOTHETICAL 69.9 KDA PROTEIN.
Analysis	Method	64	WUblastx.	WUblastx.	blastx.14	WUblastx.	2		WUblastx.	blastx.14			WUblastx.	4	WUblastx.	WUblastx.	WUblastx.	49		blastx.2	WUblastx.	WUblastx.
SEQ ID	М <u>о</u> Х		416	35	36	418			37	38			419		39	40	42			420	43	44
Contig	:O:		692291	769555	1352278	684272			834358	1352276			667830		635514	845965	639009			383592	625916	843036
cDNA	Clone ID		HANGG89	HAPOM49	HAPPW30	HAPPW30			HAPUC89	HATAC53			HATAC53		HATBR65	HATDF29	HAUAI83			HAUAI83	HBAFJ33	HBAFV19

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Ę	То	73	701	589	226	780	974	744	535	517	302	578 251	589	576	1604	448	9 <i>L</i> 6 9E8	008	245	206
Ę	From	390	492	2	158	211	1009	983	86	566	93	255	191	241	912	137	991	99	144	77
Score/	Percent Identity	41.2	48%	92%	78%.	100%	83%	65%	78%	61%	64%	82% 64%	78%	33%	74%	%19	61% 64%	91%	30.1	%6L
PFam/NR	Accession Number	PF01758	096ЕР9		AAH17488		AAK55521		Q969E3	Q924A4		Q9D6W7	Q9D6W7	gb AAC82473.1	pir H83138 H831 38	pir G83138 G831 38	Ф9Н728	pir S14350 C1HU QA	PF01391	Q9H2L.7
	PFam/NR Description	PFAM: Sodium Bile acid symporter family	(Q96EP9) Unknown (protein for IMAGE:3502817)	(Fragment).	(AAH17488) Hypothetical 22.4 kDa protein (Fragment)		(AAK55521) PR00764.		(Q969E3) Urocortin III (Stresscopin).	(Q924A4) Urocortin III.		(Q9D6W7) 2310047N01RIK PROTEIN.	(Q9D6W7) 2310047N01RIK PROTEIN.	(AF106518) sialomucin CD164 [Homo sapiens]	probable ATP-binding component of ABC transporter PA4064 [imported] - Pseudomonas aeruginosa (strain PAO1)	hypothetical protein PA4063 [imported] - Pseudomonas aeruginosa (strain PAO1)	(Q9H728) CDNA: FLJ21463 FIS, CLONE COL04765.	complement subcomponent C1q chain A precursor [validated] - human	PFAM: Collagen triple helix repeat (20 copies)	(Q9H2L7) DC33.
Analysis	Method	HMMER 2.1.1	WUblastx.	64	WUblastx.	64	WUblastx.	64	WUblastx.	WUblastx.	64	blastx.14	WUblastx.	blastx.2	WUblastx. 64	WUblastx. 64	WUblastx.	WUblastx.	HMMER 2.1.1	WUblastx.
SEQ ID	NO: X	421			423		49		20	424		51	425	426	52	427	53	54	428	
Contig	ID:	1045580			859602		514418		963208	672711		1352386	961712	892924	1130660	544980	561723	1125802	899397	
cDNA	Clone ID	HBCPB32			HBGBA69		HBIAE26		HBIMB51	HBIMB51		HBINS58	HBINS58	HBINS58	нвлтоо	HBJID05	HBJJU28	HBJNC59	HBJNC59	

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NT To		786	862	1008	820	1005	1122	1647	1648	53 928	286 773	781	1185 1320 781	1979	1079
NT From		409	64	4	497	898	298	1294	1295	9	134 453	308	1093 1264 188	1785	1002
Score/ Percent Identity		250.2	%16	%66	62%	19	%66	100%	%001	100%	%66 %001	123.9	96% 47% 98%	81%	51%
PFam/NR Accession Number		PF00386	gb AAD32626.1 AF135157_1	AAL36460	S8XN6Ò	PF00808	AAH07642	Q9H0K7	emb CAB66692. 1[Q9H8M7	DSR5_HUMAN	PF00153	Q9 HC61	Q96FR3	Q9H743
PFam/NR Description		PFAM: C1q domain	(AF135157) complement C1q A chain precursor [Homo sapiens]	(AAL36460) POB1.	(Q9NX85) CDNA FLJ20378 FIS, CLONE KAIA0536.	PFAM: Histone-like transcription factor (CBF/NF-Y) and archaeal histone	(AAH07642) Unknown (protein for IMAGE:3534358) (Fra	(Q9H0K7) HYPOTHETICAL 12.4 KDA PROTEIN (UNKNOWN) (PROTEIN FOR MGC:303	(AL.136758) hypothetical protein [Homo sapiens]	(Q9H8M7) CDNA FLJ13397 FIS, CLONE PLACE1001351.	(P57054) DOWN SYNDROME CRITICAL REGION PROTEIN 5 (DOWN SYND	PFAM: Mitochondrial carrier proteins	(Q9HC61) MITOCHONDRIAL UNCOUPLING PROTEIN 5 SHORT FORM WITH INSERTION	(Q96FR3) Unknown (protein for MGC:18083).	(Q9H743) CDNA: FLJ21394 FIS, CLONE COL03536.
Analysis Method	. 64	HMMER 2.1.1	blastx.2	WUblastx. 64	WUblastx. 64	HMMER 2.1.1	WUblastx.	WUblastx.	blastx.2	WUblastx.	WUblastx.	HMMER 2.1.1	WUblastx. 64	WUblastx.	WUblastx.
SEQ ID NO: X		429		23	85	09		61	430	62	63	64		59	99
Contig ID:		202207		842802	625923	634016		728432	494346	612796	634967	748245	·	1143407	425212
cDNA Clone ID		HBJNC59		HBXFL29	HCACU58	HCE2F54		HCE3G69	нсезсе9	HCE5F43	HCEEA88	нсеғв69		нсеғв80	HCEGR33

																		-		
TO To	1492 993	293	544	730	407	717	186	481	751	409 806	307	637	178	683	163	94	509	4	720	1167
NT From	1379	6	467	695	201	601	596	224	107	161 408	8	440	020	588	8	∞	444	318	355	946
Score/ Percent Identity	42% 58%	84%	%08	75%	2602	79%	%16	32	94%	100%	47%	42%	100%	100%	39%	36%	45%	<i>%LL</i>	36%	51%
PFam/NR Accession Number		09Н310	O9H310		000111	(2)	pir 140767 140767	PF00047	060487	060487	DVL2_MOUSE		abl A C 57877 11	Bolovojas				pir D83454 D834 54	Q92WW6	
PFam/NR Description		(Q9H310) RH TYPE B GLYCOPROTEIN.	(O9H310) RH TYPE B GLYCOPROTEIN.		(OODITI) DD O1545	(Q) [10] [NO1546.	catalase (EC 1.11.1.6) - Campylobacter jejuni	PFAM: Immunoglobulin domain	(060487) EPITHELIAL V-LIKE ANTIGEN PRECURSOR (EPITHELIAL V-LIKE ANTIG	(060487) EPITHELIAL V-LIKE ANTIGEN PRECURSOR (EPITHELIAL V-LIKE ANTIG	(Q60838) SEGMENT POLARITY PROTEIN	DISHEVELLED HOMOLOG DVL-2	similar to Dul 1 moduat anadad by ConBonk Accession	Number 1				conserved hypothetical protein PA1527 [imported] - Pseudomonas aeruginosa (strain PAO1)	(Q92WW6) Putative sensor histidine kinase protein.	
Analysis Method	5 2	WUblastx.	WUblastx.	4	11/17 11.10.04	W COLASIA.	WUblastx.	HMMER 2.1.1	WUblastx.	WUblastx.	WUblastx.	64	110.00.0	UIASIA.2				WUblastx. 64	blastx.14	
SEQ ID NO: X		<i>L</i> 9	432	1	0,7	ŝ	69	72		436	73		727	5				75	9/	
Contig ID:		941941	893535		020073	2433/0	820989	637547		589445	1134974		1045100	7010401				707833	1352416	
cDNA Clone ID		HCEWE17	HCEWE17		OCHINACOL	ncewezo	HCGMD59	HCNSM70		HCNSM70	HCOOS80		11000000	nccocse				HCWDS72	HCWEB58	

WU 03/03800														PC I/C	J502/C	70277
TO To	933 335 828	651	915	559	829	419	663	532	939	1284	1428	1415	1442	3081	1391	2891
NT From	853 264 757	442	379	350	419	538	710	708	76	277	175	723	180	259	228	69
Score/ Percent Identity	55% 41% 37%	40.4	36%	41.6	39%	77%	26%	63%	100%	81%	91%	296.3	%66	94%	613.6	%66
PFam/NR Accession Number		PF00672	pir A87396 A873 96	PF00672	gb AAA81939.1	Q9NX85			Q9UPI3	Q9Y5Y5	TMS5_HUMAN	PF00089	TMSS_HUMAN	Q9UKY2	PF01433	Q9UKY2
PFam/NR Description		PFAM: Domain found in bacterial signal proteins	sensor histidine kinase [imported] - Caulobacter crescentus	PFAM: Domain found in bacterial signal proteins	sensor-like protein [Coxiella burnetii]	(Q9NX85) CDNA FLJ20378 FIS, CLONE KAIA0536.				(Q9Y5Y5) PEROXISOMAL BIOGENESIS FACTOR 16.	(Q9H3S3) TRANSMEMBRANE PROTEASE, SERINE 5 (EC 3.4.21) (SP	PFAM: Trypsin	(Q9H3S3) TRANSMEMBRANE PROTEASE, SERINE 5 (EC 3.4.21) (SP	(Q9UKY2) ADIPOCYTE-DERIVED LEUCINE AMINOPEPTIDASE.	PFAM: Peptidase family M1	(Q9UKY2) ADIPOCYTE-DERIVED LEUCINE AMINOPEPTIDASE.
Analysis Method.		HIMMER 2.1.1	WUblastx.	HIMMER 2.1.1	blastx	WUblastx.	8		WUblastx. 64	WUblastx.	WUblastx.	HMMER 2.1.1	WUblastx. 64	WUblastx. 64	HMMER 2.1.1	WUblastx. 64
SEQ NO: NO:		439		440		11			62	80	81	441		82	442	
Contig ID:		1115089		889268		553621			610869	499233	902513	812764		1062783	866429	
cDNA Clone ID		HCWEB58		HCWEB58		HCWKC15			HCYBG92	нонев60	HDHMA45	HDHMA45		HDPBA28	HDPBA28	

cDNA	Contig	SEQ ID	Analysis		PFam/NR	Score/	Ę	Ę
Clone ID	D:	NO:	Method	PFam/NR Description	Accession Number	Percent Identity	From	To
HDPCL63	8006101	83		(Q9Y519) HYPOTHETICAL 42.3 KDA PROTEIN.	Q9Y519	%66	14	835
HDPCL63	847045	443	WUblastx. 64	(Q9Y519) HYPOTHETICAL 42.3 KDA PROTEIN.	Q9Y519	%L6	7	730
HDPCY37	669288	58	HMMER 2.1.1	PFAM: Glycosyl hydrolase family 47	PF01532	627.5	199	1521
			WUblastx. 64	(Q9H886) CDNA FLJ13869 FIS, CLONE THYRO1001287, WEAKLY SIMILAR TO MAN	988Н6О	%76	9/	1809
HDPCY37	604114	445	HMMER 2.1.1	PFAM: Glycosyl hydrolase family 47	PF01532	324	199	834
HDPFB02	808868	98	WUblastx.	(Q9BXR1) COSTIMULATORY MOLECULE.	Q9BXR1	%86	146	499
			Z			97%	877	1749
						%/6 16%	568	070
HDPFB02	1056541	446	HIMMER 2.1.1	PFAM: Immunoglobulin domain	PF00047	53.2	610	804
			blastx.2	(AF302102) costimulatory molecule [Homo sapiens]	gb AAK15438.1	93%	139	1086
НDРFB02	997408	447	HMMER 2.1.1	PFAM: Immunoglobulin domain	PF00047	26.9	305	562
			blastx.2	(AF289028) transmembrane protein B7-H2 ICOS ligand [Homo sapiens]	gb AAG01176.1 AF289028_1	%06	566	1123
нDРFF39	288867	<i>L</i> 8	WUblastx.	(096005) CLEFT LIP AND PALATE TRANSMEMBRANE PROTEIN 1.	900960	100% 100%	26 E	29
HDPGT01	771583	68	WUblastx.	(Q9Y2B3) LCAT-LIKE PROTEIN (LLPL).	О9Ү2ВЗ	100%	8 264	262
HDPJF37	704487	16	WUblastx.	(Q9BSQ8) UNKNOWN (PROTEIN FOR	Q9BSQ8	94%	105	650
	_		43	IMAGE:3510191) (FRAGMENT).		36% 93%	158	718
нррлм30	879325	92	WUblastx.	(094759) LONG TRANSIENT RECEPTOR	TRL2_HUMAN	%66	17	1633

		SEQ				7	T.	£1.4
Clone ID	Contig ID:	a ö ×	Analysis Method	PFam/NR Description	Framink Accession Number	Score/ Percent Identity	From	To
			8	POTENTIAL CHANNEL 2 (LTRPC				:
HDPJM30	603517	448	WUblastx.	(094759) LONG TRANSIENT RECEPTOR	TRL2_HUMAN	% 68	416	1312
			64	POTENTIAL CHANNEL 2 (LIRPC		96% 98%	3/8 1	378
HDPMM88	972734	66	HIMMER 2.1.1	PFAM: E1-E2 ATPase	PF00122	31	475	543
			WUblastx.	(P98198) POTENTIAL PHOSPHOLIPID. TRANSPORTING ATPASE ID (FC	ATID_HUMAN	%CE %99	106	2907
HDPMM88	906121	449	blastx.2	(AF038007) FIC1 [Homo sapiens]	gb AAC63461.1	62%	3	467
HDPMIM88	874074	452	blastx.2	(AF038007) FIC1 [Homo sapiens]	gb AAC63461.1	26%	1023	13
HDPOE32	897276	94	WUblastx. 64	(Q9BW48) MY047 PROTEIN.	Q9BW48	%86	64	345
НДРОН06	683371	95	HMMER 2.1.1	PFAM: Uncharacterized membrane protein family	PF01554	8.06	255	965
			WUblastx. 64	(Q96FL8) Hypothetical 61.9 kDa protein.	096FL8	%66	81	226
HDPOJ08	731863	96	WUblastx. 64	(Q9H7X1) CDNA FL114153 FIS, CLONE NT2RM1000092, WEAKLY SIMILAR TO MUL	Q9H7X1	84% 30% 99%	524 315 12	904 479 524
HDPPN86	1037893	16	WUblastx. 64	(Q9BVN4) HYPOTHETICAL 59.4 KDA PROTEIN.	Q9BVN4	77% 100% 97% 47%	5063 919 1942 4983 4611	5194 1308 2175 5045 4799
HDPSB18	1043263	86	WUblastx.	(Q9H5R3) CDNA: FLJ23147 FIS, CLONE LNG09295.	Q9Н5R3	70%	3363	3163
HDPSH53	1309174	66	WUblastx. 64	(Q9H257) CASPASE RECRUITMENT DOMAIN PROTEIN 9.	Q9H257	%001 100%	1011 262	1184 426
HDPSH53	1040056	459	WUblastx.	(Q9H257) CASPASE RECRUITMENT DOMAIN	Q9H257	100%	1131	1184

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NT To	1114	1114 1668 1744	1005	1440	1344	1647	2088	389	1328	1991	1332	357	2450	678 620	1149	870	1134	1382
NT From	1010	227 1078 1664	844	0/	613	544	1924	327	1242	1344	1096	13	201	637	382	402	259	183
Score/ Percent Identity	65% 92%	90% 98% 100%	38.9	% 56	30.2	%6L	85%	26%	44%	52%	27%	45%	% 66	%E8 %0\$	%89	44.3	%£9	100%
PFam/NR Accession Number		Q9BR97	PF00047	Q9Y286	PF00501	Q9BTY5							Q9H747	pir T43490 T434 90	658090	PF00027	658090	Q9BTV4
PFam/NR Description	PROTEIN 9.	(Q9BR97) UNKNOWN (PROTEIN FOR MGC:10763).	PFAM: Immunoglobulin domain	(Q9Y286) QA79 MEMBRANE PROTEIN, ALLELIC VARIANT AIRM-1B PRECURSOR.	PFAM: AMP-binding enzyme	(Q9BTYS) UNKNOWN (PROTEIN FOR MGC:4365).							(Q9H747) CDNA: FLJ21347 FIS, CLONE COL02724.	hypothetical protein DKFZp434A139.1 - human (fragments)		PFAM: Cyclic nucleotide-binding domain	(060859) NEUROPATHY TARGET ESTERASE.	(Q9BTV4) UNKNOWN (PROTEIN FOR MGC:3222).
Analysis Method	64	WUblastx. 64	HMMER 2.1.1	WUblastx. 64	HIMMER 2.1.1	WUblastx.	2						WUblastx.	WUblastx.	WUblastx.	HMMER 2.1.1	WUblastx.	WUblastx.
SEQ × Sign		461	102		103								104	105	106	469		107
Contig ID:		689129	812737		1036997								992925	879048	1309175	834692		972757
cDNA Clone ID		HDPSP01	HDPUW68		HDPVW11								HDPWN93	HDPXY01	нронроз	нронроз		HDTBD53

'n		369	718	2137 2169	1709	1721	118	1089	481	2427	805	866 106	1285	1492	270	146 985	189
Z —	To						_		ļ	_		8	<u> </u>		2		
Į,	From	0/	9	65 2131	1611	1623	14	343	116	808	281	321 71	1359	1524	1	48 269	103
Score/	Percent Identity	46%	38%	%69 %66	100%	100%	100%	84%	86.4	<i>%</i> 96	73%	31% 66%	26%	%89 8%	80%	24% 87%	44%
PFam/NR	Accession Number	Q9D513	Q9D513	Q9BT94	pir S04970 S0497 0	pir S04970 S0497 0	Q9UJU8		PF01490	69NZN8	CAD13327	9AD060	AAH07609		Q9WVT0		О9D390
	PFam/NR Description	(Q9D5J3) 4930432K09RIK PROTEIN.	(Q9D513) 4930432K09RIK PROTEIN.	(Q9BT94) UNKNOWN (PROTEIN FOR MGC:10848).	calcium-binding protein (clone pMP41) - mouse (fragment)	calcium-binding protein (clone pMP41) - mouse (fragment)	(Q9UJU8) JM24 PROTEIN (FRAGMENT).	-	PFAM: Transmembrane amino acid transporter protein	(Q9NZN8) NOT2P (CCR4-NOT TRANSCRIPTION COMPLEX, SUBUNIT 2).	(CAD13327) BA382H24.3 (multiple PDZ domain protein)	(Q9UGV6) BK445C9.3 (HIGH-MOBILITY GROUP (NONHISTONE CHROMOSOMAL) PROT	(AAH07609) Similar to hypothetical protein PRO1722.		(Q9WVT0) SEVEN TRANSMEMBRANE RECEPTOR.		(Q9D390) 6330503C03RIK PROTEIN.
Analysis	Method	\$ WUblastx.	WUblastx.	WUblastx.	WUblastx.	WUblastx. 64	WUblastx.	49	HMMER	WUblastx.	WUblastx.	WUblastx.	WUblastx.	64	WUblastx.	2	WUblastx.
SEQ ID	NO: X	108	471	109	110	472	111		475	113	115	116	117		119		120
Contig	ID:	1307742	543618	785879	1306984	879009	1043391		892317	619852	396139	740750	570903		847060		834913
cDNA	Clone ID	HDTBP04	HDTBP04	HDTBV77	ното023	нотод23	HDTFE17		HDTFE17	HE2DE47	HE2FV03	HE2NV57	HE2PH36		HE8DS15		HE8UB86

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L	То	707	467	638	653	393	405	474	1293 639 937	397	944 77	425	722 669	1042	1102
Ţ	From	432	51	360	213	148	229	529	1036 592 635	62	513 9	109	672 1	365	35
Score/	Percent Identity	61%	100%	39.7	87%	36%	24	36%	100% 100% 99%	64%	88% 65%	47%	94% 69%	130.8	95%
PFam/NR	Accession Number		AAH00573	PF00031	Q9H4G1	pir A46717 A467 17	PF01114	pir A46717 A467 17	О 9ВОМ3	Q9NP84	о об ден с	оэнвиз .	09Ү6F6	PF01762	Q9C0J1
	PFam/NR Description		(AAH00573) HSPC163 protein.	PFAM: Cystatin domain	(Q9H4G1) BA218C14.1 (NOVEL CYSTATIN FAMILY MEMBER).	colipase precursor, pancreatic - dog	PFAM: Colipase	colipase precursor, pancreatic - dog	(Q9BQM3) D1842G6.1.1 (NOVEL PROTEIN) (FRAGMENT).	(Q9NP84) TYPE I TRANSMENMBRANE PROTEIN PRECURSOR (TYPE I TRANSMEMBRAN	(Q9QZH5) PUTATIVE PHOSPHATE/PHOSPHOENOLPYRUVATE TRANSLOCATOR.	(Q9HBN2) HYPOTHETICAL 15.8 KDA PROTEIN.	(Q9Y6F6) JAW1-RELATED PROTEIN MRVIIA LONG ISOFORM.	PFAM: Galactosyltransferase	(Q9C0JI) BETA-1,3-N-ACETYLGLUCOSAMINYLTRANSFERASE BGN-T4.
Analysis	Method		WUblastx. 64	HMMER 2.1.1	WUblastx. 64	WUblastx. 64	HIMIMER 2.1.1	WUblastx. 64	WUblastx. 64	WUblastx.	WUblastx. 64	WUblastx.	WUblastx.	HMMER 2.1.1	WUblastx. 64
SEQ ID	» X		123	124		126	478		127	480	133	134	135	136	
Contig			701802	777843		885637	769649		603533	884824	566712	534142	490697	579993	
cDNA	Clone ID		невел18	HEEAQ11		HEGAN94	HEGAN94		НЕОМQ63	недссэз	нгавноз	HFAEF57	HFAMB72	н н н н н н н н н н н н н н н н н н н	

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Z	Ę	204	410	1145	919	182	458	307	411	1485	1397	1533	1434	1533	866	1846	1791	1026	1134	1601	940	934	1789	1708	1690	941	932	953
Į	From	4	249	066	842	102	423	369	497	1384	1230	1444	1390	1471	1201	2034	2009	1223	1223	1786	993	1059	2022	2040	1935	1126	1141	1144
Score/	Percent Identity	94%	92%	100%	92%	%96	%16	47%	75%	63%	54%	42%	%99	20%	%69	%09	%09	23%	40%	32%	44%	26%	63%	35%	32%	36%	44%	42%
PFam/NR	Accession Number	Q9NYC6	pir 178556 178556	AAK95397				AAK55521		Q9HAD8					060448													
	PFam/NR Description	(Q9NYC6) NEURONAL SPECIFIC TRANSCRIPTION FACTOR DATI.		(AAK95397) Selenoprotein SelM.				(AAK55521) PRO0764.		(Q9HAD8) CDNA FLJ11786 FIS, CLONE	HEMBA1006036.				(060448) NEURONAL THREAD PROTEIN AD7C-	NTP.				-								
Analysis	Method	WUblastx. 64	WUblastx. 64	WUblastx.	64	-		WUblastx.	64	WUblastx.	64				WUblastx.	2												
SEQ	ÿ×	137	139	140				141	-	142					144													
Contig	Ä	411345	513669	1043350				532060		889515					629193											-		
CDNA	Clone ID	HFCEB37	HFGAD82	HFIIZ70				HFIUR10		HFKET18					HFPA071													

									т								
NT To	1150	1697 996	1831	068	1895	1830	298	229	+77	1574	1649	689	665	1082	1015	140 1147	722
NT From	1275	1834	161	741	225	169	161	23	120	168	93	162	6	1378	5	72 134	213
Score/ Percent Identity	39%	26% 64% %	%96	46.3	% 66	94%	%98	100%	21.70	769.9	% 66	%LS	%15	%LS	81%	%96 %8L	184.6
PFam/NR Accession Number			095970	PF01463	095970	gb AAC99316.1		О9Н8Р0		PF00065	ACHG_MOUSE	095662	Q9CW46	Q9N083	рензну (AAH06833	PF00378
PFam/NR Description			(095970) LEUCINE-RICH GLIOMA-INACTIVATED PROTEIN PRECURSOR.	PFAM: Leucine rich repeat C-terminal domain	(095970) LEUCINE-RICH GLIOMA-INACTIVATED PROTEIN PRECURSOR.	(AF055636) leucine-rich glioma-inactivated protein	precursor [Homo sapiens]	(Q9H8PO) CDNA FLJ13352 FIS, CLONE	UVARCIU02103, WEARLI SIMILAR IU 3-U		(P04760) ACETYLCHOLINE RECEPTOR PROTEIN, GAMMA CHAIN PRECUR	(095662) POT. ORF VI (FRAGMENT).	(Q9CW46) 1300006N24RIK PROTEIN (FRAGMENT).	(Q9N083) UNNAMED PORTEIN PRODUCT.	(Q9H5H7) CDNA: FLJ23425 FIS, CLONE HEP22862.	(AAH06833) Similar to DKFZP586F1524 protein.	PFAM: Enoyl-CoA hydratase/isomerase family
Analysis Method			WUblastx. 64	HMMER 2.1.1	WUblastx.	blastx.2		WUblastx.	\$	HMMER 2.1.1	WUblastx.	WUblastx.	WUblastx.	WUblastx.	WUblastx.	WUblastx. 64	HMMER 2.1.1
SEQ ID NO: X			145	484		485		147		148		153	486	155	156	157	158
Contig ID:			1309793	835390		598723		545012		926569		745381	228025	701988	069859	422794	570262
cDNA Clone ID			HFPCX09	HFPCX09		HFPCX09		HFTBM50		HFTDL56		HFXGT26	HFXJU68	HFXJX44	HFXKT05	HGBFO79	НСВНІ35

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IN	То	962	1494	439	730	1403	1257	477	1505	749	950	441	1230	1373	114	114	536	787	887	1535	1706	250 1192	1523
Į	From	225	1387	7	482	723	736	79.	1251	537	12	644	10	1185	378	7	378	707	774	510	183	8 1127	1227
Score/	Percent Identity	%06	%0\$	21%	65%	62%	%L8	100%	78%	88%	87%	54%	%L6	100%	94%	94%	%86	%65	52%	148.9	%66	100% 38%	94%
PFam/NR	Accession Number	О9DBD3	pir T28058 T280	58			dbj BAA13385.1				dbj BAA13385.1	AAK55521	096ВН1		Q96FV2	096FV2	,	Q9P1J1		PF01546	Q96KN2	Q9BWV3	
	PFam/NR Description	(Q9DBD3) 1300017C12RIK PROTEIN.	hypothetical protein ZK858.6 - Caenorhabditis elegans				Similar to S. cerevisiae EMP70 protein precursor (S25110)	[Homo sapiens]			Similar to S. cerevisiae EMP70 protein precursor (S25110) [Homo sapiens]	(AAK55521) PRO0764.	(Q96BH1) Ring finger protein 25.		(Q96FV2) Unknown (protein for IMAGE:3945715) (Fragment)	1		(Q9P111) PRO1546.		PFAM: Peptidase family M20/M25/M40	(Q96KN2) Glutamate carboxypeptidase-like protein 2.	(Q9BWV3) PROTEIN KINASE NYD-SP15.	
Analysis	Method	WUblastx. 64	WUblastx.	64			blastx.2				blastx.2	WUblastx. 64	WUblastx.	64	WUblastx.	WUblastx.	64	WUblastx.	64	HMMER 2.1.1	WUblastx.	WUblastx.	
SEQ ID	» X		159				487				488	791	164		<i>L</i> 91	489		168		0/1		172	
Contig	ID:		837220				838602				899864	493724	669778		662329	383547		554613		695134		456466	
cDNA	Clone ID		HGBIB74				HGBIB74				HGBIB74	HHENK42	ННЕРМ33		HHGCM76	HHGCM76		HHGDW43		HHPEN62		HJABB94	

ANG	2,000	SEQ	Amolycic		DKom/NB	Sooro!	Ę	5
Clone ID	ing ing	ğ×	Method	PFam/NR Description	Accession	Percent Identity	From	To
HJABX32	487807	173	WUblastx.	(070277) RING FINGER PROTEIN.	070277	%86	463	612
			ŧ			35%	170	7 7
						37%	330	458
						20%	25	72
						35%	463	588
						34%	463	582
						38%	523	009
						31%	108	476
						84%	3	458
HJACG30	895505	174	WUblastx. 64	(Q9UM21) UDP-GLCNAC:A-1,3-D-MANNOSIDE B- 1,4-N-ACETYLGLUCOSAMINYLTRANS	Q9UM21	% 96	167	389
HJBCY35	719729	175	WUblastx.	hypothetical protein DKFZp586J0619.1 - human (fragment)	pir T08758 T087 58	%001	1	1212
HJPAD75	651337	177	WUblastx.	(Q9H5F8) CDNA: FLJ23476 FIS, CLONE HSI14935.	оэн5F8	%86	∞	232
HKABZ65	862030	179	WUblastx.	(Q96LB9) Peptidoglycan recognition protein-I-alpha precursor.	096ГВ9	90% 39%	77 137	802 541
HKABZ65	665424	492	WUblastx.	(Q96LB9) Peptidoglycan recognition protein-I-alpha precursor.	096ГВ9	99%	69 129	794 533
HKACB56	554616	180	HMMER 2.1.1	PFAM: Kazal-type serine protease inhibitor domain	PF00050	76.3	114	266
			WUblastx.	(P01001) ACROSIN INHIBITORS IIA AND IIB (BUSI-II).	IAC2_BOVIN	82%	96	266
HKACD58	552465	493	WUblastx.	(Q96BH2) Hypothetical 34.4 kDa protein.	О96ВН2	86% 87%	795	1208 724
НКАDQ91	604123	182	WUblastx.	(Q9NWC5) HYPOTHETICAL 31.7 KDA PROTEIN.	69NWC5	100%	229	1053
HKAEV06	638238	494	WUblastx.	(Q9NVA4) CDNA FLJ10846 FIS, CLONE	Q9NVA4	%96	367	459

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ANGS	Contia	SEC 1	_		PFom/NP	Score/	Ę	Į.
Clone ID	1D:	ië×	Method	PFam/NR Description	Accession	Percent Identity	From	To.
			2	NT2RP4001373.		100%	197	367
						%96	480	1541
HKAFK41	545018	184	WUblastx.	(BAB55101) CDNA FLJ14515 fis, clone	BAB55101	91%	18	371
			64	NT2RM1000800, w		%09	130	537
HKAFT66	946512	185	WUblastx.	(Q9CPS2) 4933428103RIK PROTEIN.	Q9CPS2	72%	53	61
			64			62%	82	231
						84%	274	828
HKAFT66	889258	495	blastx	(AF022985) No definition line found [Caenorhabditis	gb[AAB69975.1]	21%	292	543
				elegans]		25%	562	702
						79%	169	801
HKAFT66	904790	496	blastx.2	(AJ271091) B-ind1 protein [Homo sapiens]	emb CAB69070.	34%	12	296
						45%	298	516
HKB1E57	876571	186	HMMER 2.1.1	PFAM: Uncharacterized protein family UPF0004	PF00919	320.5	178	843
			WUblastx.	(Q9BWZS) DI118714.4 (CGI-05 PROTEIN (LOCS1654) SIMILAR TO RAT CDK5 AC	Q9BWZ5	%66	1	879
HKB1E57	654871	497	WUblastx.	(Q9BVG6) SIMILAR TO CGI-05 PROTEIN.	Q9BVG6	%06	<i>8L</i>	167
HKFBC53	701893	498	WUblastx.	hypothetical protein F16H11.1 - Caenorhabditis elegans	pir T16084 T160	45%	132	305
			49		84	29%	Ξ	106
						20%	82	129
						37%	999	673
						37%	293	1366
HKFBC53	513190	499	WUblastx. 64	hypothetical protein F16H11.1 - Caenorhabditis elegans	pir T16084 T160 84	35%	135	905
HKFBC53	383426	200	WUblastx.	hypothetical protein F16H11.1 - Caenorhabditis elegans	pir T16084 T160	38%	704	949
			\$		+0	27.70	133	(11)
HKGDL36	877489	189	WUblastx.	(Q9UHG2) PROSAAS PRECURSOR (GRANIN-LIKE NEUROENDOCRINE PEPTIDE PRECUR	Q9UHG2	100%	563	793 409

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NT	To	830 555	582	1013	1256	480	996	1052	295	1036	1189	562	462	1662	672	382	1142	571		989	616	955	151
IN	From	99	262	201	1107	271	532	954	468	1098	1215	332	148	1784	10	41	105	224		571	59	404	35
Score/	Percent Identity	82% 49%	28%	100%	%86	27%	26%	44%	47%	61%	77%	71%	85%	73%	47%	100%	100%	81%		95%	93%	100%	22.3
PFam/NR	Accession Number	Q9UHG2	AAL36150						Q9H7S6			Q9P059		AAG23169	Q90YM5	Q9NQW2	075477	Q9WVC2		S9N96D		бэнвzе	PF01569
	PFam/NR Description	(Q9UHG2) PROSAAS PRECURSOR (GRANIN-LIKE NEUROENDOCRINE PEPTIDE PRECUR	├—						(Q9H7S6) CDNA FLJ14310 FIS, CLONE	PLACE3000271.		(Q9P059) HSPC323 (FRAGMENT).		(AAG23169) HC6.	(Q90YM5) Organic solute transporter alpha.	(Q9NQW2) PROGRESSIVE ANKYLOSIS-LIKE	(O75477) KE04P.	(Q9WVC2) LY-6/NEUROTOXIN HOMOLOG (ADULT	MALE HIPPOCAMPUS CDNA, RIKEN	(Q96N65) CDNA FLJ31349 fis, clone MESAN2000092,	moderately similar to	(Q9HBZ6) HT005 PROTEIN.	PFAM: PAP2 superfamily
Analysis	Method	WUblastx.	WUblastx.	2					WUblastx.	49		WUblastx.	2	WUblastx.	WUblastx.	WUblastx.	WUblastx.	WUblastx.	64	WUblastx.	2	WUblastx.	HMMER 2.1.1
SEQ ID	» X	502	190						161			193		194	196	197	198	200		202		203	206
Contig	D:	704088	625956						580845			514788		581399	847396	753742	740755	460467		791828		588446	699812
cDNA	Clone ID	HKGDL36	HKISB57						HKIYP40			HKMLM11		HKMMW74	HLDOW79	HLDQR62	нгролу9	HLHFP03		HLICQ90		нгорн79	HLTHR66

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IN	To	229	740	-172	096	745	1147	420	1263	40	495	596	664	408	829	503	1006	1663	1448	1594	1331	1326	-471	846
IN	From	2	6LS	40	82	53	449	139	1003	14	19	396	503	100	470	333	38	1448	1251	1445	1260	1006	65	1
Score/	Percent Identity	93%	%08	143.1	%66	%66	78%	28%	%16	100%	83%	30%	41%	28%	26%	58%	%8 <i>L</i>	100%	100%	23%	37%	80%	71.8	%86
PFam/NR	Accession Number	Q9D4F2	9НС	PF00076	Q9NY26	Q9GZP9	Q9NRG9										Q9BY87						PF00001	H963_HUMAN
		(Q9D4F2) 4932443D16RIK PROTEIN.	(Q96DH6) Hypothetical 35.2 kDa protein.	PFAM: RNA recognition motif. (a.k.a. RRM, RBD, or RNP domain)	(Q9NY26) RT1 PROTEIN (SIMILAR TO ZINC/IRON REGULATED TRANSPORTER-LIK	(Q9GZP9) F-LAN-1 (HYPOTHETICAL TRANSMEMBRANE PROTEIN SBBI53).	(Q9NRG9) GL003 (ADRACALIN) (AAAS PROTEIN)	(UNKNOWN) (PROTEIN FOR MGC:									(Q9BY87) PROACROSIN BINDING PROTEIN SP32	PRECURSOR.					PFAM: 7 transmembrane receptor (rhodopsin family)	(O14626) PROBABLE G PROTEIN-COUPLED
Analysis	Method	WUblastx.	WUblastx. 64	HMMER 2.1.1	WUblastx. 64	WUblastx. 64	WUblastx.	45	_								WUblastx.	64	•				HMMER 2.1.1	WUblastx.
OES	ÿ×		207	504	208	500	210										211						S 0S	
Contig	D:		1087335	1047690	629552	653513	587270										658702						423998	
cDNA	Clone ID		HLTIP94	HLTIP94	HLWAA17	HLWAD77	HLWA022										HLWAY54						HLYAZ61	

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NT To		175	417	294	1023	920	440	737	483	9	100	696	327	1100	315	318	1061	866	1242	1132	538	665	1350
NT From		152	394	115	82	372	84	36	899	65	483	34	127	315	76	109	870	765	1111	1067	5/9	820	157
Score/ Percent Identity		2001	87% 96%	78%	%66	%6L	100%	95%	32%	20%	49%	93%	57.7	100%	100%	38%	27%	35%	62%	63%	25%	53%	%68
PFam/NR Accession Number		pir T47139 T471	39	Q9EPE8	Q96QY4	Q96QY4		AAL32175	062658			О9Н651	PF00595	6N9X6Q							О9Н728		901060
PFam/NR Description	RECEPTOR H963.	hypothetical protein DKFZp761P2414.1 - human		(Q9EPE8) LOW-DENSITY LIPOPROTEIN RECEPTOR-RELATED PROTEIN 9.	(Q96QY4) BA134O15.1 (similar to citrate lyase) (Fragment).	(Q96QY4) BA134015.1 (similar to citrate lyase)	(Fragment).	(AAL32175) Chromosome 17 open reading frame 26.	(062658) LINE-1 ELEMENT ORF2.			(Q9H651) CDNA: FLJ22604 FIS, CLONE HSI04630 (BBP-LIKE PROTEIN 2).	PFAM: PDZ domain (Also known as DHR or GLGF).	(Q9Y6N9) ANTIGEN NY-CO-38.	-						(Q9H728) CDNA: FLJ21463 FIS, CLONE COL04765.		(Q9D1Q6) 1110001E24RIK PROTEIN.
Analysis Method	64	WUblastx.	4	WUblastx.	blastx.14	WUblastx.	64	WUblastx. 64	WUblastx.	2		WUblastx. 64	HMMER 2.1.1	WUblastx.	64						WUblastx.	64	WUblastx.
SEQ ID NO: X		215		506	217	207		218	221			222	225								227		228
Contig ID:		561941		467053	1352406	1049263		635301	636035			560775	603201								562776		1309723
cDNA Clone ID		HMADK33		HMADU73	HMAMI15	HMAMI15		HMCFY13	HIMECK83			HMEED18	HMIAL37								HMMAH60		HMQDT36

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TN of		-265	1409	1497	1019 805	350	378 490	1225	1053	947	450	1205	226	226	1442	1438
NT From		92	192	1189	1075	781	609 609	1341	1244	1171	10	1342	14	14	42	545
Score/ Percent Identity		59.8	93%	%99	39%	83%	<i>%LL</i> %19	%09 %99	26%	64%	100%	%L9	%86	% 86	55%	%85
PFam/NR Accession Number		PF00085	Q9D1Q6	Q9H743	Q14713	095662	emb CAA26920. 1	Q9H743		Q9N083	095070	Q96AZ2	Q9NYF4	Q9NYF4	Q9D624	AAH19452
PFam/NR Description		PFAM: Thioredoxin	(Q9D1Q6) 1110001E24RIK PROTEIN.	(Q9H743) CDNA: FLJ21394 FIS, CLONE COL03536.	(Q14713) POT. ORF V.	(095662) POT. ORF VI (FRAGMENT).	pot. ORF VI [Homo sapiens]	(Q9H743) CDNA: FLJ21394 FIS, CLONE COL03536.		(Q9N083) UNNAMED PORTEIN PRODUCT.	(O95070) 54TMP.	(Q96AZ2) Similar to hypothetical protein FLJ21463.	(Q9NYF4) PUTATIVE ZINC FINGER PROTEIN.	(Q9NYF4) PUTATIVE ZINC FINGER PROTEIN.	(Q9D624) 1200003C23RIK PROTEIN.	(AAH19452) Hypothetical 49.0 kDa protein.
Analysis Method	64	HMMER 2.1.1	WUblastx.	WUblastx. 64	WUblastx.	WUblastx.	blastx.2	WUblastx.	5	WUblastx.	WUblastx.	WUblastx.	WUblastx.	WUblastx.	WUblastx.	WUblastx.
SEQ ID NO: X		208		229	230	232	512	233		234	235	237	238	513	239	514
Contig ID:		424085		973996	560229	1127691	1028961	799540		588447	639203	562063	825421	490495	1308287	794987
cDNA Clone ID		нморт36		HMSDL37	HMSFI26	HMSHS36	HMSHS36	HMSKC04		HMTAD67	HMVBS81	HMWFT65	HMWFY10	HMWFY10	HMWGY65	HMWGY65

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FN oT	122	1674	1553	552	921	713	894	498	625	917	792	915	791	595	552	462	839	786	150	992	544	1206	154	1195	200	168	711	261
NT From	0	1543	1398	334	949	645	844	331	353	828	721	781	558	401	283	379	486	145	7	516	149	1096	11	173	243	13	646	13
Score/ Percent	76%	79%	75%	%9L	26%	26%	52%	73%	26%	20%	70%	48%	20%	35%	31%	20%	61%	92%	%09	94%	91%	29%	95%	100%	%86	33%	40%	%96
PFam/NR Accession	Number	O9P195	,	060448														Q96F65		Q96F65				095400	Q96AA3			
PFam/NR Description		(O9P195) PRO1722.		(060448) NEURONAL THREAD PROTEIN AD7C-	NTP.													(Q96F65) Similar to RIKEN cDNA 0610031106 gene		⊢				(095400) CD2 CYTOPLASMIC DOMAIN BINDING PROTEIN (CD2 ANTIGEN (CYTOPLA	(Q96AA3) Putative endoplasmic reticulum multispan	transmembrane prote		
Analysis Method		WUblastx.	2	WUblastx.	42													WUblastx.	2	WUblastx.	64			WUblastx.	WUblastx.	49		
SEQ Sign	K	261		262														264		520				265	266			
Contig ID:		895462		843488														1310821		796807				570877	545534			
cDNA Clone ID		HNHNB29		HNHOD46														HNTB126		HNTBI26				HNTBIS7	HNTBL27			

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NT To	815	1499	737	1303	1303	1232	1232	311	341 918	1252	1161	872 477	892	539	776
NT From	288	204	318	290	83	336	192	192	316	1214	514	813	136	288	144
Score/ Percent Identity	189.8	85%	162.2	619	81%	496.2	%96	22.3	71% 87%	%88 88%	92%	100% 96%	93%	95.1	%86
PFam/NR Accession Number	PF00092	MTN3_HUMAN	PF00092	PF00026	pir A2 <i>577</i> 1 KHH UD	PF00026	pir A25771 KHIH UD	PF00112	BAB22302	Q9Y386		Q96BI3	69NWM8	PF00254	Q9NWM8
PFam/NR Description	PFAM: von Willebrand factor type A domain	(O15232) MATRILIN-3 PRECURSOR.	PFAM: von Willebrand factor type A domain	PFAM: Eukaryotic aspartyl protease	cathepsin D (EC 3.4.23.5) precursor [validated] - human	PFAM: Eukaryotic aspartyl protease	cathepsin D (EC 3.4.23.5) precursor [validated] - human	PFAM: Papain family cysteine protease	(BAB22302) Adult male kidney cDNA, RIKEN full-lengt	(Q9Y386) CGI-78 PROTEIN.		(Q96B13) Hypothetical 29.0 kDa protein.	(Q9NWM8) CDNA FLJ20731 FIS, CLONE HEP10272 (HYPOTHETICAL 24.2 KDA PRO	PFAM: FKBP-type peptidyl-prolyl cis-trans isomerases	(Q9NWM8) CDNA FLJ20731 FIS, CLONE HEP10272 (HYPOTHETICAL 24.2 KDA PRO
Analysis Method	HMMER 2.1.1	WUblastx.	HMMER 2.1.1	HMMER 2.1.1	WUblastx.	HIMIMER 2.1.1	WUblastx.	HMMER 2.1.1	WUblastx.	WUblastx. 64		WUblastx. 64	WUblastx.	HMMER 2.1.1	WUblastx.
SEQ ID NO:	525		526	275		531		277		278		536	280	537	
Contig ID:	968616		906694	911180		892291		931871		895880		902295	1299928	457167	
cDNA Clone ID	ноғмQ33		НОЕМОЗЗ	HOFMT75		HOFMT75		ноғос73		носск63		HOGCK63	HONAH29	HONAH29	

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NT To	585 496	489	593 544	-1719	1927	-20	3408	154	806	757	402 722 508	934 890 122 872	1415 256
NT From	436 41	100	468 143	-733	56	216	208	95	498	128	127 507 401	617 633 24 570	1317 155
Score/ Percent Identity	66% 57%	211.5	85% 86%	697.3	100%	76.2	81%	%96	48%	93%	88% 95% 97%	49% 33% 51% 35%	33% 51%
PFam/NR Accession Number	CAC37795	PF00856	Q9D8Y9	PF01747	pir JW0087 JW00 87	PF01562	ATS1_MOUSE	О9ВW19	Q96NR6	Q9CQS3	Q9CQS3	060448	
PFam/NR Description	(CAC37795) H-l(3)mbt-like protein.	PFAM: SET domain	(Q9D8Y9) 1810018L05RIK PROTEIN.	PFAM: ATP-sulfurylase	3'-phosphoadenosine-5'-phosphosulfate synthetase - human	PFAM: Reprolysin family propeptide	 		(Q96NR6) CDNA FLJ30278 fis, clone BRACE2002755.	(Q9CQS3) 1110018M03RIK PROTEIN.	(Q9CQS3) 1110018M03RIK PROTEIN.	(060448) NEURONAL THREAD PROTEIN AD7C- NTP.	
Analysis Method	WUblastx.	HMMER 2.1.1	WUblastx.	HMMER 2.1.1	WUblastx. 64	HMMER 2.1.1	WUblastx. 64	WUblastx. 64	WUblastx.	WUblastx. 64	WUblastx. 64	WUblastx. 64	
SEQ ID NO:	538	539	283	284		285		286	289	291	543	292	
Contig ID:	858338	857453	854234	614040		429229		411080	520202	1310868	590741	685699	
cDNA Clone ID	НООВ182	НООВ182	HOSDI25	HOSFD58		ноисо17		HPBCU51	HPEAD79	HPIBO15	HPB015	HPJB133	

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	Ę	To	234	256	146	942	999	099	1017	1068	336	957	1254	364 570	231	1026	884	511	310
	L	From	154	41	3	886	163	157	70	490	124	157	94	2 112	103	1100	1132	14	221 325
	Score/	Percent Identity	%65	34%	20%	47%	100%	100%	83%	51%	95%	336.4	%L6	38% 92%	%98 %98	64% 67%	34%	40%	63% 68%
	PFam/NR	Accession Number					Q9NP77	Q9NP77	AAH08720		70X16D	PF00481	О9НАҮ8	AAK33100	Q9HD20	Q9H728	pir B41925 E419	Q9BVS2	О9НА75
		PFam/NR Description					(Q9NP77) CDNA FLJ10947 FIS, CLONE PLACE1000066, WEAKLY SIMILAR TO SSU	(Q9NP77) CDNA FLJ10947 FIS, CLONE PLACE1000066, WEAKLY SIMILAR TO SSU	(AAH08720) Unknown (protein for MGC:8447).		(Q91XD7) Unknown (protein for MGC:18896).	PFAM: Protein phosphatase 2C	(Q9HAY8) SER/THR PROTEIN PHOSPHATASE TYPE 2C BETA 2 ISOFORM (PROTEIN	(AAK33100) Aminophospholipid-transporting ATPase.	(Q9HD20) CGI:152 PROTEIN.	(Q9H728) CDNA: FLJ21463 FIS, CLONE COL04765.	hypothetical protein 3 - human	(Q9BVS2) UNKNOWN (PROTEIN FOR IMAGE:3451448) (FRAGMENT).	(Q9HA75) CDNA FLJ12122 FIS, CLONE MAMMA1000129.
	Analysis	Method					WUblastx.	WUblastx.	WUblastx.	64	WUblastx. 64	HMMER 2.1.1	WUblastx. 64	WUblastx. 64	WUblastx. 64	WUblastx. 64	WUblastx.	WUblastx.	WUblastx. 64
CEO	200	ÿ×					294	547	548		549	296		297	298	302	303	304	305
	Contig	ä					846357	639118	844216		484735	829136		526310	634353	722246	585702	658717	882176
	cDNA	Clone ID					HPMDK28	HPMDK28	HPRAL78		HPRAL78	HPRBC80		HPRSB76	HPTVX32	HPWDJ42	HPZAB47	HRAAB15	HRABA80

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NT To		1701	1784	1741	1562	1126	730	579	740	-326	744	2155	633	171	961	962	478	926
NT From		793	1686	1604	999	146	825	623	75	-30	79	338	589	356	122	48	299	200
Score/ Percent Identity		70%	%09	23%	47%	%89	62%	53%	%66	92	100%	100%	73%	85%	100%	100%	54.4	88%
PFam/NR Accession Number		Q9CRM1			dbj BAB32018.1	Q9CZY7	Q9P195		Q9Y6B0	PF00254	Q96DA4	Q96F18	О951.L0	pir T42734 T427 34	pir T45062 T450 62	pir T45062 T450 62	PF01391	Q9BXJ2
PFam/NR Description		(Q9CRM1) 2610001E17RIK PROTEIN (FRAGMENT).			(AK020169) putative [Mus musculus]	(Q9CZY7) 2610307008RIK PROTEIN.	(Q9P195) PRO1722.		(Q9Y6B0) FK506-BINDING PROTEIN.	PFAM: FKBP-type peptidyl-prolyl cis-trans isomerases	(Q96DA4) FK506-binding protein.	(Q96FI8) Unknown (protein for MGC:9160).	(Q95LL0) Hypothetical 11.3 kDa protein.	cytoplasmic linker protein CLP-115 - rat	hypothetical protein c316G12.3 [imported] - human	hypothetical protein c316G12.3 [imported] - human	PFAM: Collagen triple helix repeat (20 copies)	(Q9BXJ2) COMPLEMENT-CIQ TUMOR NECROSIS
Analysis Method	64	WUblastx.	49		blastx.2	WUblastx.	WUblastx.	\$	WUblastx.	HMMER 2.1.1	lastx.	WUblastx. 64	WUblastx.	WUblastx.	WUblastx.	WUblastx.	HMMER 2.1.1	WUblastx.
SEQ ID NO: X		573			574	329	332		335	580		582	339	341	343	583	584	
Contig ID:		1074734			872570	5/09/9	467397		1306702	602258		845666	413246	898965	1056317	281098	456551	
cDNA Clone ID		HSKDA27	-		HSKDA27	HSKGN81	HSNAD72		НЅQЕО84	НЅОЕО84		HSSGD52	HSUBW09	HSVBU91	HSYBG37	HSYBG37	HSZAF47	

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NT To	397 394	397	394	412	959	585	952	488	1161	-193	231	719	700	706	952	796	1056	613	-105	614
NT From	107 344	344	353	92	408	490	548	84	319	86-	19	276	224	200	110	41	91	188	35	504
Score/ Percent Identity	62% 58%	20%	27%	93%	79%	78%	%16	99%	72%	20	100%	33%	94%	48.5	94%	%88 %	%L6	91%	31.7	100%
PFam/NR Accession Number				Q96A28		Q96A28			Q9D4I2	PF00188	90T96D			PF00047	AAG49022	AAH20905	Q9NPE6	CRES_HUMAN	PF00031	CRES_HUMAN
PFam/NR Description	FACTOR-RELATED PROTEIN.			(Q96A28) CD84-H1 (CD2 FAMILY 10).		(Q96A28) CD84-H1 (CD2 FAMILY 10).			(Q9D412) 4932408F18RIK PROTEIN.	PFAM: SCP-like extracellular protein	(Q96L06) Similar to RIKEN cDNA 1700011E04 gene.			PFAM: Immunoglobulin domain	(AAG49022) Junctional adhesion molecule 2.	(AAH20905) Hypothetical 28.5 kDa protein.	(Q9NPE6) DJ309K20.2 (ACROSOMAL PROTEIN ACR55 (SIMILAR TO RAT SPERM AN	(060676) CYSTATIN-RELATED EPIDIDYMAL SPERMATOGENIC PROTEIN	PFAM: Cystatin domain	(060676) CYSTATIN-RELATED EPIDIDYMAL
Analysis Method	64			WUblastx.	8	WUblastx.	2		WUblastx. 64	HIMMER 2.1.1	WUblastx.	2		HMMER 2.1.1	WUblastx. 64	WUblastx.	WUblastx.	WUblastx. 64	HMMER 2.1.1	WUblastx.
SEQ NO:				345		585			346	290				349		351	352	353	295	
Contig ID:				753289		457172			1018291	519372				206980		908143	600394	722254	423009	
cDNA Clone ID				HTADX17		HTADX17			HTAEE28	HTEDY42				HTEEB42		HTEGI42	нтениз1	нтени93	нтени93	

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To T		552	528	395	268	691	455	2495	423	1016	2503	482 217	501	869	619	315	448 707 474
NT From		187	22	33	23	533	3	30	40	423	911	360 14	908	12	2	193	404 567 433
Score/ Percent	Identity	78%	%18	34%	% £6	62.1	98%	94%	78%	94%	86%	100% 100%	%69	% 06	84%	73%	93% 100% 71%
PFam/NR Accession	Number		AAH20029	Q9J183	075295	PF00909	Q9UBD6	Q9UIX6	9XIN6O	•		Q9NV11	Ф9Н728	Q9D7G6	Q9D7G6	6ST96D	О9Н8Р2
PFam/NR Description		SPERMATOGENIC PROTEIN	(AAH20029) Hypothetical 39.4 kDa protein.	(Q9J183) EPCS26 (PLAC1) (PLACENTAL SPECIFIC PROTEIN 1).	(075295) R27328_2.	PFAM: Ammonium Transporter Family	(Q9UBD6) RH TYPE C GLYCOPROTEIN (TUMOR-	(Q9UJX6) ANAPHASE-PROMOTING COMPLEX SUBUNIT 2.	(09UJX6) ANAPHASE-PROMOTING COMPLEX	SUBUNIT 2.		(Q9NV11) CDNA FLJ11004 FIS, CLONE PLACE1002941.	(Q9H728) CDNA: FLJ21463 FIS, CLONE COL04765.	(Q9D7G6) 2310009N05RIK PROTEIN.	(Q9D7G6) 2310009N05RIK PROTEIN.	(Q96LS9) CDNA FLJ25101 fis, clone CBR01328.	(Q9H8P2) CDNA FLJ13348 FIS, CLONE OVARC1002127, WEAKLY SIMILAR TO SOD
Analysis Method		64	WUblastx. 64	WUblastx.	WUblastx.	HMMER 2.1.1	WUblastx.	WUblastx.	WUblastx.	49		WUblastx.	WUblastx.	WUblastx.	WUblastx.	WUblastx.	WUblastx. 64
SEQ NO:	×		355	356	357	359		360	597			361	362	865	599	364	366
Contig ID:			836072	847090	834931	706618		1040047	873355			519329	634852	791409	608317	1046341	526021
cDNA Clone ID			HTELP17	HTELS08	HTEPG70	HTJMA95		HTJML75	HTJML75			HTLAA40	HTLEP53	HTLFE57	HTLFE57	HTLIV19	нторк73

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NT To	189 519 401	1718 1650	1033	813 999	1387	2577	742	099	807	500	530	895 714	1592	819
NT From	4 418 21	1641 1507	794	172 787	302	2191	356	127	199	120	192	968 968	1488	7
Score/ Percent Identity	43% 61% 80%	96%	97.6	77% 92%	95%	100%	100%	81.5	85%	55.9	87%	70% 52%	82%	%66
PFam/NR Accession Number		67AN6Ò	PF00789	Q9CXR7	Q96S93 [°]	095880	095880	PF00822	CLD2_HUMAN	PF00822	CLD2_HUMAN	pir S22049 S2204 9	095014	Q96A54
PFam/NR Description		(Q9NVL9) CDNA FLJ 10649 FIS, CLONE NT2RP2005835, WEAKLY SIMILAR TO SHP	PFAM: UBX domain	(Q9CXR7) 3110023E09RIK PROTEIN.	(Q96S93) Hypothetical 41.7 kDa protein.	(095880) UNKNOWN.	(095880) UNKNOWN.	PFAM: PMP-22/EMP/MP20/Claudin family	(P57739) CLAUDIN-2.	PFAM: PMP-22/EMP/MP20/Claudin family	(P57739) CLAUDIN-2.	reverse transcriptase-related protein - rabbit (fragment)	(095014) WUGSC:H_D10855D21.2 PROTEIN.	(Q96A54) Similar to CGI-45 protein (Hypothetical 42.6 kDa protein).
Analysis Method		WUblastx.	HMMER 2.1.1	WUblastx. 64	WUblastx.	WUblastx.	WUblastx.	HMMER 2.1.1	WUblastx.	HMMER 2.1.1	WUblastx. 64	WUblastx.	WUblastx. 64	WUblastx. 64
SEQ NO:		368	109	698	£09	370	909	371		909		374	375	376
Contig ID:		1028538	848200	1317835	581435	854941	266683	919916		895024		1008159	429618	695765
cDNA Clone ID		HTOHM15	HTOHM15	HTPBW79	HTPBW79	HTPCS72	HTPCS72	нтрін83		нтрін83		HTTBS64	HTWCT03	HTXDW56

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L	То	565 1760	1498	327	641	563	479	462	1300	597	571	370	1338	710	316	1069	1622	1666	459	219	700	453		813	453
IN	From	470 564	1397	7	330	144	462	55	131	520	200	152	1039	597	134	281	1566	1067	-	43	317	346		229	346
Score/	Percent Identity	%66 %001	58%	22%	33%	50%	100%	93%	82%	30%	33%	29%	100%	34%	28%	%66	42%	100%	%LL	26%	100%	62.8		%59	37.8
PFam/NR	Accession Number	д 9вкно	Q96NR6	09VH80		Q9ES75	6ZN96O		Q96AA2							AAH08361			AAH08361			PF01699		Q9HC58	PF01699
	PFam/NR Description	(Q9BRH0) SIMILAR TO DKFZP727C091 PROTEIN.	(Q96NR6) CDNA FLJ30278 fis, clone BRACE2002755.	(O9VH80) CG16908 PROTEIN.		(Q9ES75) PROLINE-RICH ACIDIC PROTEIN.	(096NZ9) Proline-rich acidic protein.		(O96AA2) Obscurin.							(AAH08361) F-box only protein 7.			(AAH08361) F-box only protein 7.			PFAM: Sodium/calcium exchanger protein		(Q9HC58) SODIUM/CALCIUM EXCHANGER NCKX3.	PFAM: Sodium/calcium exchanger protein
Analysis	Method	WUblastx.	WUblastx.	WIlhlastx	49	WUblastx.	WUblastx.	64	WUblastx.	64						WUblastx.	64		WUblastx.	64		HIMMER	2.1.1	WUblastx.	HMMER 2.1.1
SEQ ID	ÿ×	377	378	610	2	612	613	;	383							614	· ·		615			387			617
Contig	ğ. E	603918	838288	161695		1300737	603538		694590	}						883176			655372			838626			833089
cDNA	Clone ID	HTXJM03	HTX0N32	HI IDR789		HUKAH51	HI IKAHS1		HIKBT29							HUSXS50			HUSXS50				HWAAD63		HWAAD63

W U U3/U38(,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,	
F of	453 793	596 453	<i>11</i> 3	785	1050	982	576	433	295	93 16	133 68	1517	694	613	1182	1800
NT From	217 533	453 319	336	207	958	215	94	104	35	134	330 148	1663	131	53	2007	169
Score/ Percent Identity	45% 41%	45% 31%	113.7	43%	83%	88%	100%	170.2	100%	78% 78%	27%	26%	100%	92%	224.2	30% 30%
PFam/NR Accession Number	gb AAF25808.1 AF177984_1		PF01699	gb AAB88884.1	Q9Y2C2	:	BAB55294	PF00255	BAB55294	AAH20829	оммо	Q9N083	075915	gb AAC64360.1	PF00028	AAK51616
PFam/NR Description	(AF177984) potassium-dependent sodium-calcium exchanger NCKX1 [Gallus gallus]		PFAM: Sodium/calcium exchanger protein	(AF025664) Na-Ca+K exchanger [Bos taurus]	(Q9Y2C2) DERMATAN/CHONDROITIN SULFATE 2-	SOLI'OI INAINSI ERASE.	(BAB55294) CDNA FLJ14777 fis, clone NT2RP4000259, w	PFAM: Glutathione peroxidases	(BAB55294) CDNA FLJ14777 fis, clone NT2RP4000259, w	(AAH20829) Hypothetical 6.2 kDa protein.	(Q96MIM0) CDNA FLJ32172 fis, clone PLACE6000555.	(Q9N083) UNNAMED PORTEIN PRODUCT.	(075915) JWA PROTEIN (HSPC127) (VITAMIN A RESPONSIVE, CYTOSKELETON RE	(AF070523) JWA protein [Homo sapiens]	PFAM: Cadherin domain	(AAK51616) Protocadherin-beta10.
Analysis Method	blastx.2		HMMER 2.1.1	blastx.2	WUblastx.	5	WUblastx.	HMMER 2.1.1	WUblastx.	WUblastx.	WUblastx.	WUblastx.	WUblastx.	blastx.2	HMMER 2.1.1	WUblastx.
SEQ NO.			618		390		391	619		392	620	394	395	622	968	
Contig ID:			793875		836469		1093347	886210		846382	646977	799427	805642	801943	762842	
cDNA Clone ID			HWAAD63		HWBAR88		HWBCB89	HWBCB89		HWBCP79	HWBCP79	HWBFX31	НWНН1.34	HWHHL34	НWНQS55	

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NT To	2563 1632	455	623	1464	1130	1397	1130	1397	1130	1397
NT From	1817 520	147	81	1147	924	468	924	540	924	468
Score/ Percent Identity	80% 28%	49.3	%86	62%	<i>L</i> 9	100%	29	92%	<i>2</i> 9	100%
PFam/NR Accession Number		PF01940	AAH08596	Q9NX17	PF00047	Q9BX67	PF00047	gb AAK27221.1 AF356518_1	PF00047	. 69вх67
PFam/NR Description		PFAM: Integral membrane protein	(AAH08596) Unknown (protein for MGC:16985).	(Q9NX17) CDNA FLJ20489 FIS, CLONE KAT08285.	PFAM: Immunoglobulin domain	(Q9BX67) JUNCTIONAL ADHESION MOLECULE 3 PRECURSOR.	PFAM: Immunoglobulin domain	(AF356518) junctional adhesion molecule 3 precursor [Homo sapiens]	PFAM: Immunoglobulin domain	(Q9BX67) JUNCTIONAL ADHESION MOLECULE 3 PRECURSOR.
Analysis Method		HMMER 2.1.1	WUblastx.	WUblastx.	HMMER 2.1.1	WUblastx.	HMMER 2.1.1	blastx.2	HMMER 2.1.1	WUblastx.
SEQ NO:		397		398	401		624		625	
Contig ID:		793713		826754	846517		887467		878627	
cDNA Clone ID		HWLIH65		HYAAJ71	HAPSA79		HAPSA79		HAPSA79	

RACE Protocol For Recovery of Full-Length Genes

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Partial cDNA clones can be made full-length by utilizing the rapid amplification of cDNA ends (RACE) procedure described in Frohman, M.A., et al., Proc. Nat'l. Acad. Sci. USA, 85:8998-9002 (1988). A cDNA clone missing either the 5' or 3' end can be reconstructed to include the absent base pairs extending to the translational start or stop codon, respectively. In some cases, cDNAs are missing the start codon of translation, therefor. The following briefly describes a modification of this original 5' RACE procedure. Poly A+ or total RNA is reverse transcribed with Superscript II (Gibco/BRL) and an antisense or complementary primer specific to the cDNA sequence. The primer is removed from the reaction with a Microcon Concentrator (Amicon). The first-strand cDNA is then tailed with dATP and terminal deoxynucleotide transferase (Gibco/BRL). Thus, an anchor sequence is produced which is needed for PCR amplification. The second strand is synthesized from the dA-tail in PCR buffer, Taq DNA polymerase (Perkin-Elmer Cetus), an oligo-dT primer containing three adjacent restriction sites (XhoI, SalI and ClaI) at the 5' end and a primer containing just these restriction sites. This double-stranded cDNA is PCR amplified for 40 cycles with the same primers as well as a nested cDNA-specific antisense primer. The PCR products are size-separated on an ethidium bromideagarose gel and the region of gel containing cDNA products the predicted size of missing proteincoding DNA is removed. cDNA is purified from the agarose with the Magic PCR Prep kit (Promega), restriction digested with XhoI or SaII, and ligated to a plasmid such as pBluescript SKII (Stratagene) at XhoI and EcoRV sites. This DNA is transformed into bacteria and the plasmid clones sequenced to identify the correct protein-coding inserts. Correct 5' ends are confirmed by comparing this sequence with the putatively identified homologue and overlap with the partial cDNA clone. Similar methods known in the art and/or commercial kits are used to amplify and recover 3' ends.

Several quality-controlled kits are commercially available for purchase. Similar reagents and methods to those above are supplied in kit form from Gibco/BRL for both 5' and 3' RACE for recovery of full length genes. A second kit is available from Clontech which is a modification of a related technique, SLIC (single-stranded ligation to single-stranded cDNA), developed by Dumas et al., Nucleic Acids Res., 19:5227-32 (1991). The major differences in procedure are that the RNA is alkaline hydrolyzed after reverse transcription and RNA ligase is used to join a restriction site-containing anchor primer to the first-strand cDNA. This obviates the necessity for the dA-tailing reaction which results in a polyT stretch that is difficult to sequence past.

An alternative to generating 5' or 3' cDNA from RNA is to use cDNA library double-stranded DNA. An asymmetric PCR-amplified antisense cDNA strand is synthesized with an antisense cDNA-specific primer and a plasmid-anchored primer. These primers are removed and a symmetric PCR reaction is performed with a nested cDNA-specific antisense primer and the plasmid-anchored primer.

RNA Ligase Protocol For Generating The 5' or 3' End Sequences To Obtain Full Length Genes

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Once a gene of interest is identified, several methods are available for the identification of the 5' or 3' portions of the gene which may not be present in the original cDNA plasmid. These methods include, but are not limited to, filter probing, clone enrichment using specific probes and protocols similar and identical to 5' and 3' RACE. While the full length gene may be present in the library and can be identified by probing, a useful method for generating the 5' or 3' end is to use the existing sequence information from the original cDNA to generate the missing information. A method similar to 5'_RACE is available for generating the missing 5' end of a desired full-length gene. (This method was published by Fromont-Racine et al., Nucleic Acids Res., 21(7):1683-1684 (1993)). Briefly, a specific RNA oligonucleotide is ligated to the 5' ends of a population of RNA presumably containing full-length gene RNA transcript and a primer set containing a primer specific to the ligated RNA oligonucleotide and a primer specific to a known sequence of the gene of interest, is used to PCR amplify the 5' portion of the desired full length gene which may then be sequenced and used to generate the full length gene. This method starts with total RNA isolated from the desired source, poly A RNA may be used but is not a prerequisite for this procedure. The RNA preparation may then be treated with phosphatase if necessary to eliminate 5' phosphate groups on degraded or damaged RNA which may interfere with the later RNA ligase step. The phosphatase if used is then inactivated and the RNA is treated with tobacco acid pyrophosphatase in order to remove the cap structure present at the 5' ends of messenger RNAs. This reaction leaves a 5' phosphate group at the 5' end of the cap cleaved RNA which can then be ligated to an RNA oligonucleotide using T4 RNA ligase. This modified RNA preparation can then be used as a template for first strand cDNA synthesis using a gene specific oligonucleotide. The first strand synthesis reaction can then be used as a template for PCR amplification of the desired 5' end using a primer specific to the ligated RNA oligonucleotide and a primer specific to the known sequence of the gene of interest. The resultant product is then sequenced and analyzed to confirm that the 5' end sequence belongs to the relevant gene.

The present invention also relates to vectors or plasmids which include such DNA sequences, as well as the use of the DNA sequences. The material deposited with the ATCC (e.g., as described in columns 2 and 3 of Table 1A, and/or as set forth in Table 1B, Table 6, or Table 7) is a mixture of cDNA clones derived from a variety of human tissue and cloned in either a plasmid vector or a phage vector, as described, for example, in Table 1A and Table 7. These deposits are referred to as "the deposits" herein. The tissues from which some of the clones were derived are listed in Table 7, and the vector in which the corresponding cDNA is contained is also indicated in Table 7. The deposited material includes cDNA clones corresponding to SEQ ID NO:X described, for example, in Table 1A

and/or Table 1B (ATCC Deposit No:Z). A clone which is isolatable from the ATCC Deposits by use of a sequence listed as SEQ ID NO:X, may include the entire coding region of a human gene or in other cases such clone may include a substantial portion of the coding region of a human gene. Furthermore, although the sequence listing may in some instances list only a portion of the DNA sequence in a clone included in the ATCC Deposits, it is well within the ability of one skilled in the art to sequence the DNA included in a clone contained in the ATCC Deposits by use of a sequence (or portion thereof) described in, for example Tables 1A and/or Table 1B or Table 2, by procedures hereinafter further described, and others apparent to those skilled in the art.

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Also provided in Table 1A and Table 7 is the name of the vector which contains the cDNA clone. Each vector is routinely used in the art. The following additional information is provided for convenience.

Vectors Lambda Zap (U.S. Patent Nos. 5,128,256 and 5,286,636), Uni-Zap XR (U.S. Patent Nos. 5,128, 256 and 5,286,636), Zap Express (U.S. Patent Nos. 5,128,256 and 5,286,636), pBluescript (pBS) (Short, J. M. et al., Nucleic Acids Res. 16:7583-7600 (1988); Alting-Mees, M. A. and Short, J. M., Nucleic Acids Res. 17:9494 (1989)) and pBK (Alting-Mees, M. A. et al., Strategies 5:58-61 (1992)) are commercially available from Stratagene Cloning Systems, Inc., 11011 N. Torrey Pines Road, La Jolla, CA, 92037. pBS contains an ampicillin resistance gene and pBK contains a neomycin resistance gene. Phagemid pBS may be excised from the Lambda Zap and Uni-Zap XR vectors, and phagemid pBK may be excised from the Zap Express vector. Both phagemids may be transformed into E. coli strain XL-1 Blue, also available from Stratagene.

Vectors pSport1, pCMVSport 1.0, pCMVSport 2.0 and pCMVSport 3.0, were obtained from Life Technologies, Inc., P. O. Box 6009, Gaithersburg, MD 20897. All Sport vectors contain an ampicillin resistance gene and may be transformed into *E. coli* strain DH10B, also available from Life Technologies. See, for instance, Gruber, C. E., et al., *Focus* 15:59- (1993). Vector lafmid BA (Bento Soares, Columbia University, New York, NY) contains an ampicillin resistance gene and can be transformed into *E. coli* strain XL-1 Blue. Vector pCR®2.1, which is available from Invitrogen, 1600 Faraday Avenue, Carlsbad, CA 92008, contains an ampicillin resistance gene and may be transformed into *E. coli* strain DH10B, available from Life Technologies. See, for instance, Clark, J. M., *Nuc. Acids Res.* 16:9677-9686 (1988) and Mead, D. et al., Bio/Technology 9: (1991).

The present invention also relates to the genes corresponding to SEQ ID NO:X, SEQ ID NO:Y, and/or the deposited clone (ATCC Deposit No:Z). The corresponding gene can be isolated in accordance with known methods using the sequence information disclosed herein. Such methods include preparing probes or primers from the disclosed sequence and identifying or amplifying the corresponding gene from appropriate sources of genomic material.

Also provided in the present invention are allelic variants, orthologs, and/or species homologs. Procedures known in the art can be used to obtain full-length genes, allelic variants, splice variants, full-length coding portions, orthologs, and/or species homologs of genes corresponding to SEQ ID NO:X or the complement thereof, polypeptides encoded by genes corresponding to SEQ ID NO:X or the complement thereof, and/or the cDNA contained in ATCC Deposit No:Z, using information from the sequences disclosed herein or the clones deposited with the ATCC. For example, allelic variants and/or species homologs may be isolated and identified by making suitable probes or primers from the sequences provided herein and screening a suitable nucleic acid source for allelic variants and/or the desired homologue.

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The polypeptides of the invention can be prepared in any suitable manner. Such polypeptides include isolated naturally occurring polypeptides, recombinantly produced polypeptides, synthetically produced polypeptides, or polypeptides produced by a combination of these methods. Means for preparing such polypeptides are well understood in the art.

The polypeptides may be in the form of the secreted protein, including the mature form, or may be a part of a larger protein, such as a fusion protein (see below). It is often advantageous to include an additional amino acid sequence which contains secretory or leader sequences, pro-sequences, sequences which aid in purification, such as multiple histidine residues, or an additional sequence for stability during recombinant production.

The polypeptides of the present invention are preferably provided in an isolated form, and preferably are substantially purified. A recombinantly produced version of a polypeptide, including the secreted polypeptide, can be substantially purified using techniques described herein or otherwise known in the art, such as, for example, by the one-step method described in Smith and Johnson, Gene 67:31-40 (1988). Polypeptides of the invention also can be purified from natural, synthetic or recombinant sources using techniques described herein or otherwise known in the art, such as, for example, antibodies of the invention raised against the polypeptides of the present invention in methods which are well known in the art.

The present invention provides a polynucleotide comprising, or alternatively consisting of, the nucleic acid sequence of SEQ ID NO:X, and/or the cDNA sequence contained in ATCC Deposit No:Z. The present invention also provides a polypeptide comprising, or alternatively, consisting of, the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X or a complement thereof, a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z, and/or the polypeptide sequence encoded by a nucleotide sequence in SEQ ID NO:B as defined in column 6 of Table 1C. Polynucleotides encoding a polypeptide comprising, or alternatively consisting of the polypeptide sequence of SEQ ID NO:Y, a polypeptide encoded by SEQ ID NO:X, a polypeptide encoded by the

cDNA contained in ATCC Deposit No:Z, and/or a polypeptide sequence encoded by a nucleotide sequence in SEQ ID NO:B as defined in column 6 of Table 1C are also encompassed by the invention. The present invention further encompasses a polynucleotide comprising, or alternatively consisting of, the complement of the nucleic acid sequence of SEQ ID NO:X, a nucleic acid sequence encoding a polypeptide encoded by the complement of the nucleic acid sequence of SEQ ID NO:X, and/or the cDNA contained in ATCC Deposit No:Z.

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Moreover, representative examples of polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the sequences delineated in Table 1C column 6, or any combination thereof. Additional, representative examples of polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the complementary strand(s) of the sequences delineated in Table 1C column 6, or any combination thereof. In further embodiments, the above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in Table 1C, column 6, and have a nucleic acid sequence which is different from that of the BAC fragment having the sequence disclosed in SEQ ID NO:B (see Table 1C, column 5). In additional embodiments, the above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in Table 1C, column 6, and have a nucleic acid sequence which is different from that published for the BAC clone identified as BAC ID NO:A (see Table 1C, column 4). In additional embodiments, the above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in Table 1C, column 6, and have a nucleic acid sequence which is different from that contained in the BAC clone identified as BAC ID NO:A (see Table 1C, column 4). Polypeptides encoded by these polynucleotides, other polynucleotides that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides and polypeptides are also encompassed by the invention.

Further, representative examples of polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the sequences delineated in column 6 of Table 1C which correspond to the same Clone ID (see Table 1C, column 1), or any combination thereof. Additional, representative examples of polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the complementary strand(s) of the sequences delineated in column 6 of Table 1C which correspond to the same Clone ID (see Table 1C, column 1), or any combination thereof. In further embodiments, the above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in column 6 of Table 1C which correspond to the same Clone ID (see Table 1C, column 1)

and have a nucleic acid sequence which is different from that of the BAC fragment having the sequence disclosed in SEQ ID NO:B (see Table 1C, column 5). In additional embodiments, the above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in column 6 of Table 1C which correspond to the same Clone ID (see Table 1C, column 1) and have a nucleic acid sequence which is different from that published for the BAC clone identified as BAC ID NO:A (see Table 1C, column 4). In additional embodiments, the above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in column 6 of Table 1C which correspond to the same Clone ID (see Table 1C, column 1) and have a nucleic acid sequence which is different from that contained in the BAC clone identified as BAC ID NO:A (see Table 1C, column 4). Polypeptides encoded by these polynucleotides, other polynucleotides that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides and polypeptides are also encompassed by the invention.

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Further, representative examples of polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the sequences delineated in column 6 of Table 1C which correspond to the same contig sequence identifier SEQ ID NO:X (see Table 1C, column 2), or any combination thereof. Additional, representative examples of polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the complementary strand(s) of the sequences delineated in column 6 of Table 1C which correspond to the same contig sequence identifier SEQ ID NO:X (see Table 1C, column 2), or any combination thereof. In further embodiments, the above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in column 6 of Table 1C which correspond to the same contig sequence identifier SEQ ID NO:X (see Table 1C, column 2) and have a nucleic acid sequence which is different from that of the BAC fragment having the sequence disclosed in SEQ ID NO:B (see Table 1C, column 5). In additional embodiments, the above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in column 6 of Table 1C which correspond to the same contig sequence identifier SEQ ID NO:X (see Table 1C, column 2) and have a nucleic acid sequence which is different from that published for the BAC clone identified as BAC ID NO:A (see Table 1C, column 4). In additional embodiments, the abovedescribed polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in column 6 of Table 1C which correspond to the same contig sequence identifier SEQ ID NO:X (see Table 1C, column 2) and have a nucleic acid sequence which is different from that contained in the BAC clone identified as BAC ID NO:A (See Table 1C, column 4). Polypeptides encoded by these polynucleotides, other polynucleotides that encode these polypeptides, and antibodies that bind these

polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides and polypeptides are also encompassed by the invention.

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Moreover, representative examples of polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the sequences delineated in the same row of Table 1C column 6, or any combination thereof. Additional, representative examples of polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the complementary strand(s) of the sequences delineated in the same row of Table 1C column 6, or any combination thereof. In preferred embodiments, the polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the complementary strand(s) of the sequences delineated in the same row of Table 1C column 6, wherein sequentially delineated sequences in the table (i.e. corresponding to those exons located closest to each other) are directly contiguous in a 5' to 3' orientation. In further embodiments, above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in the same row of Table 1C, column 6, and have a nucleic acid sequence which is different from that of the BAC fragment having the sequence disclosed in SEQ ID NO:B (see Table 1C, column 5). In additional embodiments, the above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in the same row of Table 1C, column 6, and have a nucleic acid sequence which is different from that published for the BAC clone identified as BAC ID NO:A (see Table 1C, column 4). In additional embodiments, the above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated in the same row of Table 1C, column 6, and have a nucleic acid sequence which is different from that contained in the BAC clone identified as BAC ID NO:A (see Table 1C, column 4). Polypeptides encoded by these polynucleotides, other polynucleotides that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention.

In additional specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the sequences delineated in column 6 of Table 1C, and the polynucleotide sequence of SEQ ID NO:X (e.g., as defined in Table 1C, column 2) or fragments or variants thereof. Polypeptides encoded by these polynucleotides, other polynucleotides that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention.

In additional specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the sequences delineated in column 6 of Table 1C which correspond to the same Clone ID (see Table 1C, column 1), and the polynucleotide sequence of SEQ ID NO:X (e.g., as defined in Table 1A, Table 1B, or Table 1C) or

fragments or variants thereof. In preferred embodiments, the delineated sequence(s) and polynucleotide sequence of SEQ ID NO:X correspond to the same Clone ID. Polypeptides encoded by these polynucleotides, other polynucleotides that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention.

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In further specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more of the sequences delineated in the same row of column 6 of Table 1C, and the polynucleotide sequence of SEQ ID NO:X (e.g., as defined in Table 1A, Table 1B, or Table 1C) or fragments or variants thereof. In preferred embodiments, the delineated sequence(s) and polynucleotide sequence of SEQ ID NO:X correspond to the same row of column 6 of Table 1C. Polypeptides encoded by these polynucleotides, other polynucleotides that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention.

In additional specific embodiments, polynucleotides of the invention comprise, or alternatively consist of a polynucleotide sequence in which the 3' 10 polynucleotides of one of the sequences delineated in column 6 of Table 1C and the 5' 10 polynucleotides of the sequence of SEQ ID NO:X are directly contiguous. Nucleic acids which hybridize to the complement of these 20 contiguous polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or nucleic acids that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides are also encompassed by the invention.

In additional specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, a polynucleotide sequence in which the 3' 10 polynucleotides of one of the sequences delineated in column 6 of Table 1C and the 5' 10 polynucleotides of a fragment or variant of the sequence of SEQ ID NO:X are directly contiguous Nucleic acids which hybridize to the complement of these 20 contiguous polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or nucleic acids encoding these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides are also encompassed by the invention.

In specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, a polynucleotide sequence in which the 3' 10 polynucleotides of the sequence of SEQ ID NO:X and

the 5' 10 polynucleotides of the sequence of one of the sequences delineated in column 6 of Table 1C are directly contiguous. Nucleic acids which hybridize to the complement of these 20 contiguous polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or nucleic acids encoding these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides are also encompassed by the invention.

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In specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, a polynucleotide sequence in which the 3' 10 polynucleotides of a fragment or variant of the sequence of SEQ ID NO:X and the 5' 10 polynucleotides of the sequence of one of the sequences delineated in column 6 of Table 1C are directly contiguous. Nucleic acids which hybridize to the complement of these 20 contiguous polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or nucleic acids encoding these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides, are also encompassed by the invention.

In further specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, a polynucleotide sequence in which the 3' 10 polynucleotides of one of the sequences delineated in column 6 of Table 1C and the 5' 10 polynucleotides of another sequence in column 6 are directly contiguous. Nucleic acids which hybridize to the complement of these 20 contiguous polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or nucleic acids encoding these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides are also encompassed by the invention.

In specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, a polynucleotide sequence in which the 3' 10 polynucleotides of one of the sequences delineated in column 6 of Table 1C and the 5' 10 polynucleotides of another sequence in column 6 corresponding to the same Clone ID (see Table 1C, column 1) are directly contiguous. Nucleic acids which hybridize to the complement of these 20 lower stringency conditions, are also encompassed by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or

nucleic acids encoding these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides are also encompassed by the invention.

In specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, a polynucleotide sequence in which the 3' 10 polynucleotides of one sequence in column 6 corresponding to the same contig sequence identifer SEQ ID NO:X (see Table 1C, column 2) are directly contiguous. Nucleic acids which hybridize to the complement of these 20 contiguous polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or nucleic acids encoding these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides are also encompassed by the invention.

In specific embodiments, polynucleotides of the invention comprise, or alternatively consist of a polynucleotide sequence in which the 3' 10 polynucleotides of one of the sequences delineated in column 6 of Table 1C and the 5' 10 polynucleotides of another sequence in column 6 corresponding to the same row are directly contiguous. In preferred embodiments, the 3' 10 polynucleotides of one of the sequences delineated in column 6 of Table 1C is directly contiguous with the 5' 10 polynucleotides of the next sequential exon delineated in Table 1C, column 6. Nucleic acids which hybridize to the complement of these 20 contiguous polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or nucleic acids encoding these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides are also encompassed by the invention.

Table 3

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Many polynucleotide sequences, such as EST sequences, are publicly available and accessible through sequence databases and may have been publicly available prior to conception of the present invention. Preferably, such related polynucleotides are specifically excluded from the scope of the present invention. Accordingly, for each contig sequence (SEQ ID NO:X) listed in the fifth column of Table 1A and/or the fourth column of Table 1B, preferably excluded are one or more polynucleotides comprising a nucleotide sequence described by the general formula of a-b, where a is any integer between 1 and the final nucleotide minus 15 of SEQ ID NO:X, b is an integer of 15 to the final

nucleotide of SEQ ID NO:X, where both a and b correspond to the positions of nucleotide residues shown in SEQ ID NO:X, and where b is greater than or equal to a + 14. More specifically, preferably excluded are one or more polynucleotides comprising a nucleotide sequence described by the general formula of a-b, where a and b are integers as defined in columns 4 and 5, respectively, of Table 3. In specific embodiments, the polynucleotides of the invention do not consist of at least one, two, three, four, five, ten, or more of the specific polynucleotide sequences referenced by the Genbank Accession No. as disclosed in column 6 of Table 3 (including for example, published sequence in connection with a particular BAC clone). In further embodiments, preferably excluded from the invention are the specific polynucleotide sequence(s) contained in the clones corresponding to at least one, two, three, four, five, ten, or more of the available material having the accession numbers identified in the sixth column of this Table (including for example, the actual sequence contained in an identified BAC clone). In no way is this listing meant to encompass all of the sequences which may be excluded by the general formula, it is just a representative example. All references available through these accessions are hereby incorporated by reference in their entirety.

FABLE 3

	-		AI089027, AA308141, AW504673, AI684832, AA225036, AI806235, AA480904, AW084470, BE246140, AI769587, AA480993, AA936449, AI743330, AW025616, R84772, AI244944, N58917, AI085514, AA504299, AI273353, AI762989, AA100979, AA857531, AW276652, AW952845, AW440624, AI277859, R74507, AW269427, AI221905, AW016095, H72021, AI50547, H65671, T89998, AI937672, H86848, R74517, R52128, BE243519, AA224988, AA588111, T89414, AA976027, Z39380, BE869329, R48449, R72429, AA229997, AA308518, BF183288, AA229612, AI694870, AV755614, AV755613, T24832, AA229703, AA620967, AA594460, AA480981, AA480883, BF059107, AA278692, AV691613, AI197824, H65670, AA480992, AA480966, AC003070. 1.	AL537848, BE796835, BE793657, BE793638, BF968748, BE727036, BF316464, BE728420, BF314482, BE868759, BE407252, BE409490, BE276749, BE386000, BF026545, BE871737, BE384252, BG235902, BE261749, BF125396, Al961321, Al001128, Al34334, AW135558, BE729783, AA777237, AA478021, AK673734, AR875954, BF270372, Al 537847, AA232184, AA478177, BF220321, BE965762, BE276538.
	claimer Range of b	15 - 2703	15 - 459	15 - 1939
	EST Disclaimer Range of a Range	1 - 2689	1 - 445	1 - 1925
	Contig ID:	884134	544957	135222
SEQ ID	ë×	-	12	13
	cDNA Clone ID	H2CBU83	H2MAC30	H6EAB28

9,	29,		66, 72, 7, 7, 824, 48, 48, 802, 1376, 11, 11, 11, 11, 11, 11, 11, 11, 11, 1	33, 33, 33, 3,
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HDTGC73	112	635457	1 - 698	15 - 712	AW022607, AW511178, AI140427, AI971228, AI373655, AI580779, AI369886, AI190934, H40803, AI243231, AA453827, AA453746, BF446909, BE326968, AA961079, AA040716, N47998, AI819706,	
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					AW10/41/, N98819, AA048346, A1/21089, Br3/46/6, AA311809, Br331280, AW731809, Br3003, Br100374, W73034 BF110011, BF347329, BF382364, AA718927, N66559, BE832805, AA679466, A1224843,	
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	HE8UB86	HE9HY07	HE9NN84	HEBEJ18

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HEGAN94	126	885637	1 - 568	15 - 582	AJ018488, BF509739, AL157823. 9.
НЕОМО63	127	603533	1 - 1322	15 - 1336	BG026315, AW102828, AI659843, BE551400, AI640582, BE208434, BF510823, AW955647, BE669917,

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	15 - 1126	15 - 2657	15 - 2410	15 - 2131
	1 - 1112	1 - 2643	1 - 2396	1 - 2117
	119486	833079	118446 5	911180
	272	273	274	275
	HODFN71	HOEFV61	ноғмозз	HOFMT75

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HOFOC73	277	931871	1 - 1477	15 - 1491	BF110813, BF939079, AW573230, BE747230, AI760936, BF348602, AA418800, AI870845, AI420441,					

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293	294			295
HPJBK12	HPMDK28			HPRAL78

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	1 - 2529	1 - 727	1 - 789	1 - 805		1 - 309
	829136	526310	634353	526749	0	413270
	296	297	298	300	3	301
·	HPRBC80	HPRSB76	HPTVX32	HPVAB94		HPWAZ95

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15 - 1340 A A A A A A A A A A A A A A A A A A A	15 - 1676 A	15 - 1747 B	15 - 1251 A
1 - 1326	1 - 1662	1 - 1733	1 - 1237
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HPWD142	HPZAB47	HRAAB15	HRABA80

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	- 1525	1 - 2063   15
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	HRACD15	HRACI35

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	HRGBL78	HROAJ39	нковр68

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HSKHZ81	330	130710	1 - 955	15 - 969	AI814274, BF869496, AI092236, AI275399, AI970748, AW381532, BF828729, BF828779, AI355259, AA055367, AA582963, BF825322, BE158757, BE158812, BE182090, BE182079, BE140770, BF737235, BE182110, BF836665, AW238620, BF828691, BF828827, AA055699, BE711192, BE717546, BF737527, BE140771, BF094219, BE158774, BF826017, BF838537, BE939669, AC002389. 1.
HSLJG37	331	101692	1-2112	15 - 2126	AI733659, AI792379, AI003110, BF931916, AC022608, AC022608, AC022608.
HSNAD72	332	467397	1 - 847	15 - 861	W861646, A1610321, A1880774, AA829195, A1880765, AA551170, A196983; B870, AA557945, AW873417, A1635819, C06160, AV761107, BE268727, AV F574331, BG222875, BF946125, BF882222, BE068993, BF946124, AA49384 821901, BE044000, A1701898, H86399, H47461, A1338426, A1926093, AC0 AC006254.10, AL035462.21, AC018868.4, AC008747.5, AC090527.3, AI AC06254.10, AL035462.21, AC010422.7, AC010267.6, AC011609.9, AL136981.22, AL391241.21, AC010422.7, AC010267.6, AC011609.9, AL1363807.18, AL049776.3, Z98200.8, AC067722.21, AC010913.9, AC AL161656.20, AL122020.5, AL157372.18, AC067722.21, AC010913.9, AC AL136979.1, AC002404.1, AC008482.5, AL035404.20, AL136304.10, AC AL136979.16, AL031660.16, Z83844.5, AC004813.2, AL136304.10, AC AL136979.16, AL031660.16, Z83844.5, AC0025588.1, AL145212.9, AC008484.5, AC002384.4, U95742.1, AC004000.1, AL117381.32, AC AL137077.31, AL031733.3, AL45490.6, AC023790.21, AL13733.3, AL45490.6, AC025165.27, AC018711.4, AP000038.1, AL590763.1, AC004846.2, AC003041.1, AL133238.3, AL137918.4, AC007163.3, AP000555.1, AL135905.6, AL133905.6, AL133907.3, AL133933.3, AL133907.3, AL133908.4, AL138330.1, AL139383.3, AL13330.6, AL133333.3, AL13600.1, AL139333.3, AL13600.1, AL158933.2, AC006515.7, AL139997.14, AC006515.1, AC006452.4, AL1588390.17, AC006515.1, AL189997.14, AP001724.1, AC006452.4, AL1588390.17, AC0066515.1, AL189997.14, AP001724.1, AC006452.4, AL1588390.17, AC006655.1, AL189997.14, AP001724.1, AC006452.4, AL1588390.17, AC006655.1, AL189997.14, AP001724.1, AC006452.4, AL1588390.17, AC006655.1, AL189997.14, AP001724.1, AC006452.4, AL1588390.17, AC006655.1, AL189997.14, AP001724.1, AC006452.4, AL1588390.17, AC006655.1, AL189997.14, AP001724.1, AC006452.4, AL1588390.17, AC006655.1, AL189997.14, AC006522.1, AC006452.4, AL1588390.17, AC006655.1, AL189997.14, AC006522.1, AC006522.1, AC006652.1, AC006652.1, AC006652.1, AC006652.1, AC006652.1, AC006652.2, AC006652.2, AC006652.2, AC006652.2, AC006652.2, AC006652.2, AC006652.2, AC006652.2, AC006652.2, AC006652.2, AC006652.2, AC006652.2, AC006652.2, AC006652.2, AC00
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HSQEO84	335	130670 2	1-917	15 - 931	BE874396, BF210667, AV700682, AI271550, AI753504, AW082138, AV745382, AA809220, AW081079, AI879695, AV746084, AV753894, BF576504, AI879318, AI264068, AI219556, AV746036, AW973033, AA455733, AI382746, AA431230, AA548778, AV696126, BE673279, AW386283, AW608255, W78099, AA432251, AW608247, AI344174, AI344234, AF100751.1, AC009948.3, AL109755.14, Z62799.1, Z64678. 1.

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15 - 477	13 - 1930	15 - 2425	15 - 1021	15 - 1155	15 - 727	15 - 1112	15 - 1238
1 - 463	1 - 1910	1 - 2411	1 - 1007	1 - 1141	1 - 713	1 - 1098	1 - 1224
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336	337	338	339	340	341	342	343
HSQFP66	HSRF257	HSSGD52	HSUBW09	HSVAT68	HSVBU91	HSXEC75	HSYBG37

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BF770293, AA628898, A1094804, A1056500, BF820729, A1375863, BE857274, AW959629, AA905637, A1382011, A1860596, AA877674, AA513392, A1459017, W73121, A1242677, A1309547, BF819613, H16241, T70036, AA642494, AI675842, T03552, AI689235, BF919604, AI274910, AV748307, C00014, A1678921, AA993626, AI201747, AA742201, BF351125, BF770277, AW082362, BF914643, T30150, A1014513, R93245, H83130, AI538135, AI244946, A1952326, N45499, BF344569, AI685761, H05737, F20400, BF914807, A1094715, A1810996, AA320821, BF222389, H20376, H20185, H83129, T10108, AA370998, H12111, T70103, BF915044, AI800313, R47434, AI557606, AA299558, BF983106, BF347892, N47608, BF590090, AI141309, BF915129, AW385116, AA885897, BF917925, BF917920, N89795, AI797519, AA652696, AI193489, AA894705, T10109, AW189222, BF350004, BE829911, AI224610, AI423668, AA872954, AI207820, R39978, AI99899, AI640906, BE046990, AI206927, AA136304, AW614497, AI751243, AA128437, AW772433, AI640184, AW385115, BF326281, W39052, AA595730, BF088390, AA731862, BF338332, BF111399, BF770143, BE767158, AA983866, AL9302786.1, AE006467.1, AL031709.12, AK024842. 1.	AW298370, AI433823, AI239867, D62170, D61860, AF329839.1, AC007016. 5.	AA446344, AA612751, AA298785, AA298780, AA298784, AA446524, AA298781, AA381170.	AW195720, AI765273, AI817356, AI928166, AI283845, BE503396, AW081502, BE349083, BF059350, AA419437, AA758800, AW206944, AA933673, AW104261, AI627565, AI264565, AW469909, AA845240, AA332515, AL021453. 1.	AA437009, AI806582, AI040972, AA442839, AA759268, AI214390, AI799076, AA918443, AW195596, AA910234.	AA393537, A1187279, AA889534, AW002667, AA421499, AW003587, AA421468, AA709184, AW772510, AA397830.	AL522795, AA725566, AI421450, AL522796, AI199779, AA406389, AA912674, AW022835, AI952846, AI123727, BE218057, AW022646, N90730, BF846982, BF845761, AI652914, BF056970, AW020783, AI312805, AW393829, AI017553, AW39387, AW474261, AW264246, BF848293, AI366088, AI418268, T89217, AI052637, AW082343, BF221504, AW593293, AA865038, AI201753, BF091146, AI140139, AA987434, AA410345, BF846977, BF846980, AW900593, BF932982, BF932991, AW865421, AW136481, AI650503, AI432092, T89127, AA974715, AW261924, BE938414, AF255910.1, AY016009.1, AP001694.1, AP000025.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP000026.1, AP00002	AW072387, R83559, AI924465, AI364031, AW513660, BF361111, AA705541, AL162032. 1.	BF792295, AW118908, AW956740, AA805770, AA578718, AA805757, AA808355, AA805773, N29112, AI760754, AI005113, AI204164, N21153, AI004282, AW956741, AI001990, BE564602, AI538204, AI188040, AI301191, AA383104, AW182071, AI192033, BE168090, AA861920, N31710, AA887975,	AA976455, BF812960, AI249323, AIS64247, AI619607, AI961286, AI819976, AI567612, AI632033, AI554821, AI934036, AI538116, AI251434, BE964614, BF826445, AW105601, AI818980, AI926790, H89138, AI269862, AI288285, AW079075, AI280747, AW055252, AI621362, AV648430, AI590423,
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	344	345	346	347	348	349	350	351	
	HSZAF47	HTADX17	HTAEE28	HTECC05	HTEDY42	НТЕЕВ42	HTEFU65	HTEG142	

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	1 - 1884	1 - 799	1 - 1804	1 - 1636			1 - 2748
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	571200	119481	838626
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	HUVEB53	HVARW53	нwaad63

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	15 - 1440	15 - 838
	1 - 1426	1 - 824 1 - 4372
	610383	846517
	399	401
	HYBAR01	HAPSA79

## Description of Table 4

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Table 4 provides a key to the tissue/cell source identifier code disclosed in Table 1B.2, column 5. Column 1 of Table 4 provides the tissue/cell source identifier code disclosed in Table 1B.2, Column 5. Columns 2-5 provide a description of the tissue or cell source. Note that "Description" and "Tissue" sources (i.e. columns 2 and 3) having the prefix "a_" indicates organs, tissues, or cells derived from "adult" sources. Codes corresponding to diseased tissues are indicated in column 6 with the word "disease." The use of the word "disease" in column 6 is non-limiting. The tissue or cell source may be specific (e.g. a neoplasm), or may be disease-associated (e.g., a tissue sample from a normal portion of a diseased organ). Furthermore, tissues and/or cells lacking the "disease" designation may still be derived from sources directly or indirectly involved in a disease state or disorder, and therefore may have a further utility in that disease state or disorder. In numerous cases where the tissue/cell source is a library, column 7 identifies the vector used to generate the library.

Disease Organ colon cancer (9808co64R) Donor II Resting B Cells Donor II B-Cells 24 hrs. Donor II B-Cells 72hrs Blood B cells activated colon cancer 9809co14 Donor II B Cells 24hrs Donor II B Cells 72hrs Blood T cells activated Blood B cells resting Blood T cells resting cell line transformed a_mammary gland colon (9808co65R) Cell Line CAOV3 colon (9809co15) a_small intestine Blood B cells breast cancer cell line PA-1 colon cancer a_Stomach a_Prostate a Heart breast a_Liver brain colon colon cancer 9809co14 Donor II B Cells 24hrs Donor II B Cells 72hrs Donor II B-Cells 72hrs Blood B cells activated Blood T cells activated Blood B cells resting Blood T cells resting cell line transformed Donor II B-Cells 24 Donor II Resting B colon (9808co65R) a_mammary gland Cell Line CAOV3 Description colon (9809co15) a_small intestine Blood B cells cell line PA-1 breast cancer colon cancer (9808co64R) colon cancer a_Stomach a_Prostate a_Heart a_Liver breast colon brain AR024 AR027 AR054 AR025 AR041 AR052 AR042 AR053 AR026 AR031 **AR028** AR029 AR030 AR032 AR034 AR035 AR036 **AR037 AR038 AR039 AR040** AR043 AR044 AR050 AR051 **AR022** AR033 AR023 Code

TABLE 4

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
	Cells					
AR055	Heart	Heart				
AR056	Human Lung (clonetech)	Human Lung (clonetech)				
AR057	Human Mammary (clontech)	Human Mammary (clontech)				
AR058	Human Thymus (clonetech)	Human Thymus (clonetech)				
AR059	Jurkat (unstimulated)	Jurkat (unstimulated)				
AR060	Kidney	Kidney				
AR061	Liver	Liver				
AR062	Liver (Clontech)	Liver (Clontech)				
AR063	Lymphocytes chronic lymphocytic leukaemia	Lymphocytes chronic lymphocytic leukaemia				
AR064	Lymphocytes diffuse large B cell lymphoma	Lymphocytes diffuse large B cell lymphoma				
AR065	Lymphocytes follicular	Lymphocytes follicular				
AR066	normal breast	normal breast				
AR067	Normal Ovarian (4004901)	Normal Ovarian (4004901)				
AR068	Normal Ovary 9508G045	Normal Ovary 9508G045				
AR069	Normal Ovary 9701G208	Normal Ovary 9701G208		-		
AR070	Normal Ovary 9806G005	Normal Ovary 9806G005				
AR071	Ovarian Cancer	Ovarian Cancer				
AR072		Ovarian Cancer (9702G001)	:			
AR073	Ovarian Cancer (9707G029)	Ovarian Cancer (9707G029)				

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Vector																		
Disease										-	i							
Cell Line														•				
Organ																		
Tissue	Ovarian Cancer (9804G011)	Ovarian Cancer (9806G019)	Ovarian Cancer (9807G017)	Ovarian Cancer (9809G001)	ovarian cancer 15799	Ovarian Cancer 17717AID	Ovarian Cancer 4004664B1	Ovarian Cancer 4005315A1	ovarian cancer 94127303	Ovarian Cancer 96069304	Ovarian Cancer 9707G029	Ovarian Cancer 9807G045	ovarian cancer 9809G001	Ovarian Cancer 9905C032RC	Ovarian cancer 9907 C00 3rd	Prostate	Prostate (clonetech)	prostate cancer
Description	Ovarian Cancer (9804G011)	Ovarian Cancer (9806G019)	Ovarian Cancer (9807G017)	Ovarian Cancer (9809G001)	ovarian cancer 15799	Ovarian Cancer 17717AD	Ovarian Cancer 4004664B1	Ovarian Cancer 4005315A1	ovarian cancer 94127303	Ovarian Cancer 96069304	Ovarian Cancer 9707G029	Ovarian Cancer 9807G045	ovarian cancer 9809G001	Ovarian Cancer 9905C032RC	Ovarian cancer 9907 C00 3rd	Prostate	Prostate (clonetech)	prostate cancer
Code	AR074	AR075	AR076	AR077	AR078	AR079	AR080	AR081	AR082	AR083	AR084	AR085	AR086	AR087	AR088	AR089	AR090	AR091

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
AR092	prostate cancer #15176	prostate cancer #15176				
AR093	prostate cancer #15509	prostate cancer #15509				
AR094	prostate cancer #15673	prostate cancer #15673				
AR095	Small Intestine (Clontech)	Small Intestine (Clontech)				
AR096	Spleen	Spleen				
AR097	Thymus T cells	Thymus T cells activated				
000	activated	=				
AR098	Thymus T cells resting	Thymus T cells resting				
AR099	Tonsil	Tonsil				
AR100		Tonsil geminal center				
	centroblast	centroblast				
AR101	Tonsil germinal center B cell	Tonsil germinal center B cell				
AR 102	Tonsil lymph node	Tonsil lymph node				
AR103	Tonsil memory B cell	Tonsil memory B cell				
AR104	Whole Brain	Whole Brain				
AR105	Xenograft ES-2	Xenograft ES-2				
AR106	Xenograft SW626	Xenograft SW626				
AR119	001: IL-2	001: IL-2			•	
	001: П2.1	001: IL-2.1				
AR121	001: IL-2_b	001: IL-2_b				
AR124	002: Monocytes	002: Monocytes untreated				
	untreated (1hr)	(1hr)		-		
AR125	002: Monocytes	002: Monocytes untreated				
	untreated (5hrs)	(Shrs)				
AR126	002: Control.1C	002: Control.1C				
AR127	002: IL2.1C	002: IL2.1C				
AR130	003 : Placebo-treated	003 : Placebo-treated Rat				
	Rat Lacrimal Gland	Lacrimal Gland				
AR131	003: Placebo-treated	003 : Placebo-treated Rat				

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
	Rat Submandibular	Submandibular Gland				
	Gland					
AR135	004 : Monocytes	004: Monocytes untreated (5hrs)				
AR136	004 · Monocytes	004 : Monocytes untreated				
2	untreated 1hr	1hr				
AR139	005: Placebo (48hrs)	005: Placebo (48hrs)				
AR140	006: pC4 (24hrs)	006: pC4 (24hrs)				
AR141	006: pC4 (48hrs)	006: pC4 (48hrs)				
AR152	007: PHA(1hr)	007: PHA(1hr)				
AR153	007: PHA(6HRS)	007: PHA(6HRS)				
AR154	007: PMA(6hrs)	007: PMA(6hrs)				
AR155	008: 1449_#2	008: 1449_#2				
AR161	01: A - max 24	01: A - max 24				
AR162	01: A - max 26	01: A - max 26				
AR163	01: A - max 30	01: A - max 30				
AR164	01: B - max 24	01: B - max 24				
AR165	01: B - max 26	01: B - max 26				
AR166	01: B - max 30	01: B - max 30				
AR167	1449 Sample	1449 Sample				
AR168	3T3P10 1.0uM insulin	3T3P10 1.0uM insulin				
AR169	3T3P10 10nM Insulin	3T3P10 10nM Insulin				
AR170	3T3P10 10uM insulin	3T3P10 10uM insulin				
AR171	3T3P10 No Insulin	3T3P10 No Insulin		-		
AR172	3T3P4	3T3P4				
AR173	Adipose (41892)	Adipose (41892)				
	Adipose Diabetic (41611)	Adipose Diabetic (41611)				
AR175	Adipose Diabetic (41661)	Adipose Diabetic (41661)				
AR176	Adipose Diabetic	Adipose Diabetic (41689)				

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
	(41689)					
AR177	Adipose Diabetic (41706)	Adipose Diabetic (41706)				
AR178	Adipose Diabetic (42352)	Adipose Diabetic (42352)				
AR179	Adipose Diabetic (42366)	Adipose Diabetic (42366)				
AR180	Adipose Diabetic (42452)	Adipose Diabetic (42452)				
AR181	Adipose Diabetic (42491)	Adipose Diabetic (42491)				
AR182	Adipose Normal (41843)	Adipose Normal (41843)				
AR183	Adipose Normal (41893)	Adipose Normal (41893)				
AR184	Adipose Normal (42452)	Adipose Normal (42452)				
AR185	Adrenal Gland	Adrenal Gland				
AR186	Adrenal Gland + Whole Brain	Adrenal Gland + Whole Brain				
AR187	B7(1hr)+ (inverted)	B7(1hr)+ (inverted)				
AR188	Breast (18275A2B)	Breast (18275A2B)				
AR189	Breast (4004199)	Breast (4004199)				
AR190	Breast (4004399)	Breast (4004399)		-		
AR191	Breast (4004943B7)	Breast (4004943B7)				
AR192	Breast (4005570B1)	Breast (4005570B1)				
AR193	Breast Cancer (4004127A30)	Breast Cancer (4004127A30)				
AR194	Breast Cancer (400443A21)	Breast Cancer (400443A21)				
AR195	Breast Cancer (4004643A2)	Breast Cancer (4004643A2)				

Vector	,																	:
Disease																		
Cell Line							•											
Organ																		
Tissue	Breast Cancer (4004710A7)	Breast Cancer (4004943A21)	Breast Cancer (400553A2)	Breast Cancer (9805C046R)	Breast Cancer (9806C012R)	Breast Cancer (ODQ 45913)	Breast Cancer (ODQ45913)	Breast Cancer (ODQ4591B)	Colon Cancer (15663)	Colon Cancer (4005144A4)	Colon Cancer (4005413A4)	Colon Cancer (4005570B1)	Control RNA #1	Control RNA #2	Cultured Preadipocyte (blue)	Cultured Preadipocyte (Red)	Donor II B-Cells 24hrs	Donor II Resting B-Cells
Description	Breast Cancer (4004710A7)	Breast Cancer (4004943A21)	Breast Cancer (400553A2)	Breast Cancer (9805C046R)	Breast Cancer (9806C012R)	Breast Cancer (ODQ 45913)	Breast Cancer (ODQ45913)	Breast Cancer (ODQ4591B)	Colon Cancer (15663)	Colon Cancer (4005144A4)	Colon Cancer (4005413A4)	Colon Cancer (4005570B1)	Control RNA #1	Control RNA #2	Cultured Preadipocyte (blue)	Cultured Preadipocyte (Red)	Donor II B-Cells 24hrs	Donor II Resting B-
Code	AR196	AR197	AR198	AR199	AR200	AR201	AR202	AR203	AR204	AR205	AR206	AR207	AR208	AR209	AR210	AR211	AR212	AR213

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
AR214	H114EP12 10nM	H114EP12 10nM Insulin				
	IIIsuilli					
AR215	H114EP12 (10nM insulin)	H114EP12 (10nM insulin)		···-		
AR216	H114EP12 (2.6ug/ul)	H114EP12 (2.6ug/ul)				
AR217	H114EP12 (3.6ug/ul)	H114EP12 (3.6ug/ul)				
AR218	HUVEC#1	HUVEC#1				
AR219	HUVEC#2	HUVEC #2				
AR221	L6 undiff.	L6 undiff.				
AR222	L6 Undifferentiated	L6 Undifferentiated				
AR223	L6P8 + 10nM Insulin	L6P8 + 10nM Insulin				
AR224	L6P8 + HS	SH + 849′T				
AR225	L6P8 10nM Insulin	L6P8 10nM Insulin				
AR226	Liver (00-06-A007B)	Liver (00-06-A007B)				
AR227	Liver (96-02-A075)	Liver (96-02-A075)				•
AR228	Liver (96-03-A144)	Liver (96-03-A144)				
AR229	Liver (96-04-A138)	Liver (96-04-A138)				
AR230	Liver (97-10-A074B)	Liver (97-10-A074B)				
AR231	Liver (98-09-A242A)	Liver (98-09-A242A)				
AR232	Liver Diabetic (1042)	Liver Diabetic (1042)				
AR233	Liver Diabetic (41616)	Liver Diabetic (41616)				
AR234	Liver Diabetic (41955)	Liver Diabetic (41955)				
AR235	Liver Diabetic (42352R)	Liver Diabetic (42352R)				
AR236	Liver Diabetic (42366)	Liver Diabetic (42366)				
AR237	Liver Diabetic (42483)	Liver Diabetic (42483)				
AR238	Liver Diabetic (42491)	Liver Diabetic (42491)				
AR239	Liver Diabetic (99-09-	Liver Diabetic (99-09-				
$\neg$	A281A)	A281A)				
	Lung	Lung				
AR241	Lung (27270)	Lung (27270)				

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
AR242	Lung (2727Q)	Lung (2727Q)				
AR243	Lung Cancer (4005116A1)	Lung Cancer (4005116A1)				
AR244	Lung Cancer (4005121A5)	Lung Cancer (4005121A5)				
AR245	Lung Cancer (4005121A5))	Lung Cancer (4005121A5))				
AR246	Lung Cancer (4005340A4)	Lung Cancer (4005340A4)				
AR247	Mammary Gland	Mammary Gland				
AR248	Monocyte (CT)	Monocyte (CT)				
AR249	Monocyte (OCT)	Monocyte (OCT)				
AR250	Monocytes (CT)	Monocytes (CT)				
AR251	Monocytes (INFG 18 hr)	Monocytes (INFG 18 hr)				
AR252	Monocytes (INFG 18hr)	Monocytes (INFG 18hr)				
AR253	Monocytes (INFG 8-11)	Monocytes (INFG 8-11)				
AR254	Monocytes (O CT)	Monocytes (O CT)				
AR255		Muscle (91-01-A105)				
AR256	Muscle (92-04-A059)	Muscle (92-04-A059)				
AR257	Muscle (97-11-A056d)	Muscle (97-11-A056d)				
AR258	Muscle (99-06-A210A)	Muscle (99-06-A210A)				
AR259	Muscle (99-07-A203B)	Muscle (99-07-A203B)				
AR260	Muscle (99-7-A203B)	Muscle (99-7-A203B)				
AR261	Muscle Diabetic (42352R)	Muscle Diabetic (42352R)				
AR262	Muscle Diabetic (42366)	Muscle Diabetic (42366)				
AR263	NK-19 Control	NK-19 Control				
AR264	NK-19 IL Treated	NK-19 IL Treated 72hrs				

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
	72hrs					
AR265	NK-19 UK Treated 72 hrs.	NK-19 UK Treated 72 hrs.		÷	•	
AR266	Omentum Normal (94- 08-B009)	Omentum Normal (94-08- B009)				
AR267	Omentum Normal (97- 01-A039A)	Omentum Normal (97-01- A039A)				
AR268	Omentum Normal (97- 04-A114C)	Omentum Normal (97-04-A114C)				
AR269	Omentum Normal (97- 06-A117C)	Omentum Normal (97-06- A117C)				
AR270	Omentum Normal (97- 09-B004C)	Omentum Normal (97-09- B004C)				
AR271	Ovarian Cancer (17717AID)	Ovarian Cancer (17717AJD)				
AR272	Ovarian Cancer (9905C023RC)	Ovarian Cancer (9905C023RC)				
AR273	Ovarian Cancer (9905C032RC)	Ovarian Cancer (9905C032RC)				
AR274	Ovary (9508G045)	Ovary (9508G045)				
-	Ovary (9701G208)	Ovary (9701G208)				
$\neg  au$	Ovary 9806G005	Ovary 9806G005				
AR278	Fancreas Placebo	Placebo				
AR279	rIL2 Control	rIL2 Control				
AR280	RSS288L	RSS288L				
AR281	RSS288LC	RSS288LC				
AR282	Salivary Gland	Salivary Gland				
AR283	Skeletal Muscle	Skeletal Muscle				
AR284	Skeletal Muscle (91- 01-A105)	Skeletal Muscle (91-01- A105)				
AR285	Skeletal Muscle	Skeletal Muscle (42180)				

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Code	Description	Tissue	Organ	Cell Line	Disease	Vector
AR367	B-Lymphoblast	B-Lymphoblast				
AR368	B-Lymphocytes	B-Lymphocytes				
AR369	Bladder	Bladder				
AR370	Bone Marrow	Bone Marrow				
AR371	Bronchial Epithelial	Bronchial Epithelial Cell				
	Cell					
AR372	Bronchial Epithelial	Bronchial Epithelial Cells				
	Cells					
AR373	Caco-2A	Caco-2A				
AR374	Caco-2B	Caco-2B				
AR375	Caco-2C	Caco-2C				
AR376	Cardiac #1	Cardiac #1				
AR377	Cardiac #2	Cardiac #2				
AR378	Chest Muscle	Chest Muscle				
AR381	Dendritic Cell	Dendritic Cell				
AR382	Dendritic cells	Dendritic cells				
AR383	E.coli	E.coli				
AR384	Epithelial Cells	Epithelial Cells				
_	Esophagus	Esophagus				
	БРРS	FPPS				
AR387	FPPSC	FPPSC				
AR388	HepG2 Cell Line	HepG2 Cell Line				
AR389	HepG2 Cell line Buffer 1 hr.	HepG2 Cell line Buffer 1 hr.		-		
AR390	HepG2 Cell line Buffer 06 hr	HepG2 Cell line Buffer 06 hr				
AR391	HepG2 Cell line Buffer 24 hr.	HepG2 Cell line Buffer 24 hr.				
AR392	HepG2 Cell line Insulin 01 hr.	HepG2 Cell line Insulin 01 hr.				
AR393	HepG2 Cell line	HepG2 Cell line Insulin 06				

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
	Insulin 06 hr.	hr.				
AR394	HepG2 Cell line Insulin 24 hr.	HepG2 Cell line Insulin 24 hr.				
AR398	HMC-1	HMC-1				
AR399	HMCS	HMCS				
AR400	_	OSWH				
AR401	HUVEC#3	HUVEC #3				
AR402	HUVEC #4	HUVEC #4				
AR404	KIDNEY NORMAL	KIDNEY NORMAL				
AR405	KIDNEY TUMOR	KIDNEY TUMOR				
AR406	KIDNEY TUMOR					
AR407	Lymph Node	Lymph Node				
AR408		Macrophage				
AR409	Megakarioblast	Megakarioblast				
AR410	_	Monocyte				
AR411	Monocytes	Monocytes				
AR412	Myocardium	Myocardium				
AR413	Myocardium #3	Myocardium #3				
AR414	Myocardium #4	Myocardium #4		•		
AR415	Myocardium #5	Myocardium #5				
AR416	NK	NK				
AR417	NK cell	NK cell				
AR418	NK cells	NK cells				
AR419	NKYa	NKYa				
AR420	NKYa019	NKYa019				
AR421	Ovary	Ovary				
AR422	Patient #11	Patient #11				
AR423		Peripheral blood				
AR424	Primary Adipocytes	Primary Adipocytes				

Vector																			Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR		Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR
Disease		-								·																			
Cell Line									٠																				
Organ																			Heart	Spleen	Brain				Kidney	Kidney	Embryo	Gall Bladder	Gall Bladder
Tissue	Promyeloblast	RSSWT	RSSWTC	SW 480(G1)	SW 480(G2)	SW 480(G3)	SW 480(G4)	SW 480(G5)	T Lymphoblast	T Lymphocyte	T-Cell	T-Cell,	T-Cells	T-lymphoblast	Th 1	Th 2	Th1	Th2	Human Adult Heart	Human Adult Spleen	Human Cerebellum				Human Fetal Kidney	Human Fetal Kidney	Human 8 Week Old Embryo	Human Gall Bladder	Human Gall Bladder
Description	Promyeloblast	RSSWT	RSSWTC	SW 480(G1)	SW 480(G2)	SW 480(G3)	SW 480(G4)	SW 480(G5)	T Lymphoblast	T Lymphocyte	T-Cell	T-Cell,	T-Cells	T-lymphoblast	Th 1	Th 2	Th1	Th2	Human Adult Heart	Human Adult Spleen	Human Cerebellum	Whole 6 Week Old	Embryo	Human Fetal Brain	Human Fetal Kidney	Human Fetal Kidney	Human 8 Week Whole	Human Gall Bladder	Human Gall Bladder, fraction II
Code	AR425	AR427	AR428	AR429	AR430	AR431	AR432	AR433	├-	AR435	AR436	AR438		AR440	AR441	-	AR443	AR444	H0002	H0004	H0007	H0008		6000H	H0011	H0012	H0013	H0014	T

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0016	Human Greater	Human Greater Omentum	peritoneum			Uni-ZAP XR
	Omentum					
H0022	Jurkat Cells	Jurkat T-Cell Line				Lambda ZAP II
H0024	Human Fetal Lung III	Human Fetal Lung	Lung			Uni-ZAP XR
H0025	Human Adult Lymph Node	Human Adult Lymph Node	Lymph Node			Lambda ZAP II
H0026	Namalwa Cells	Namalwa B-Cell Line, EBV immortalized				Lambda ZAP II
H0030	Human Placenta					Uni-ZAP XR
H0031	Human Placenta	Human Placenta	Placenta			Uni-ZAP XR
H0032	Human Prostate	Human Prostate	Prostate			Uni-ZAP XR
H0033	Human Pituitary	Human Pituitary				Uni-ZAP XR
H0036	Human Adult Small Intestine	Human Adult Small Intestine	Small Int.			Uni-ZAP XR
H0038	Human Testes	Human Testes	Testis			Uni-ZAP XR
H0039	Human Pancreas Tumor	Human Pancreas Tumor	Pancreas		disease	Uni-ZAP XR
H0040	Human Testes Tumor	Human Testes Tumor	Testis		disease	Uni-ZAP XR
H0041	Human Fetal Bone	Human Fetal Bone	Bone			Uni-ZAP XR
H0042	Human Adult Pulmonary	Human Adult Pulmonary	Lung			Uni-ZAP XR
H0045	Human Esophagus, Cancer	Human Esophagus, cancer	Esophagus		disease	Uni-ZAP XR
H0046	Human Endometrial Tumor	Human Endometrial Tumor	Uterus	-	disease	Uni-ZAP XR
H0047	Human Fetal Liver	Human Fetal Liver	Liver			Uni-ZAP XR
H0050	Human Fetal Heart	Human Fetal Heart	Heart			Uni-ZAP XR
H0051	Human Hippocampus	Human Hippocampus	Brain			Uni-ZAP XR
H0052	Human Cerebellum	Human Cerebellum	Brain			Uni-ZAP XR
H0056	Human Umbilical Vein, Endo. remake	Human Umbilical Vein Endothelial Cells	Umbilical vein			Uni-ZAP XR
H0057	Human Fetal Spleen					Uni-ZAP XR

Human Uterine Cancer         Human Uterine Cancer         Uterus           Human Macrophage         Blood         Cell Line           Human Rin Tumor         Skin         Cell Line           Human Skin Tumor         Human Skin Tumor         Skin           Human Activated Tr-Cells         Blood         Cell Line           Human Pancreas         Human Pancreas         Pancreas           Human Infant Adrenal         Human Pancreas         Pancreas           Human Infant Adrenal         Human Pancreas         Pancreas           Human Leiomyeloid         Muscle         Cell Line           Cals (II)         Human Tell         Blood         Cell Line           Human Activated Tr         Activated Tr-Cells         Blood         Cell Line           Cells (II)         Human Thymus Tumor         Thymus         Cell Line           Human Activated Tr         Activated Tr-Cells         Skin         Blood           Human Colon         Human Thymus         Tr-Cell Lymphoma         Tr-Cell Lymphoma           Human Colon         Human Adult Heart         Human Adult Heart         Heart           Human Adult Heart         Human Adult Heart         Heart         Subbracted           Human Adult Liver         Human Whole Six Week Old         <	Code	Description	Tissue	Organ	Cell Line	Disease	Vector
Human Macrophage         Blood         Cell Line           Human Thymus         Thymus         Thymus           Human Skin Tumor         Skin         disease           Human Skin Tumor         Blood         Cell Line           Galls         Activated T-Cells         Blood         Cell Line           Galls         Human Pancreas         Pancreas         Pancreas           Human Pancreas         Human Pancreas         Pancreas           Human Pancreas         Human Pancreas         Pancreas           Human Leiomyeloid         Activated T-Cells         Blood         Cell Line           Carcinoma         Carcinoma         Carcinoma         disease           Human Cacinoma         Carcinoma         Alixea         disease           Cells (II)         Human Thymus Tumor         Thymus         disease           Edithelium (Skin)         Jurkat Cells         Skin         disease           Human Colon         Human Colon         Human Colon         Human Colon           Human Thymus         Human Thymus         T-Cell         disease           Human Thymus         Human Adult Heart         Heart         Heart           Human Adult Heart         Heart         Heart         Heart	H0059	Human Uterine Cancer		Uterus		disease	Lambda ZAP II
Human Thymus         Human Thymus         Thymus           Human Activated T-Cells         Skin         disease           Human Activated T-Cells         Activated T-Cells         Blood         Cell Line           Human Pancreas         Human Pancreas         Pancreas           Human Infant Adrenal Gland         Adrenal gland         disease           Human Infant Adrenal Gland         Adrenal gland         disease           Carcinoma         Carcinoma         Carcinoma           Human Activated T-Cells         Blood         Cell Line           Carcinoma         Activated T-Cells         Blood         Cell Line           Garcinoma         Human Petal Skin         Skin         disease           Human Petal         Human Fetal Skin         Skin         disease           Human Colon         Human Colon         Fittlelioid Sarcoma, muscle         Sk Muscle         disease           Human Thymus         Human Thymus         T-Cell Lymphoma         T-Cell Lymphoma         disease           Human Thymus         Human Adult Heart         Human Adult Heart         Heart         disease           Human Adult Heart,         Human Adult Liver,         Human Adult Liver         Liver         Human Adult Liver           Human Whole Six	H0061	Human Macrophage	Human Macrophage	Blood	Cell Line		pBluescript
Human Skin Tumor         Human Skin Tumor         Activated T-Cells         Blood         Cell Line         disease           Human Activated T-Cells         Human Pancreas         Pancreas         Pancreas         Items           Human Infant Adrenal         Human Infant Adrenal Gland         Adrenal gland         Gland         Gland           Human Leiomyeloid         Human Leiomyeloid         Muscle         Gell Line         disease           Carcinoma         Human Leiomyeloid         Muscle         Cell Line         disease           Cells (II)         Human Thymus Tumor         Thymus         disease         disease           Epithelium (Skin)         Human Fetal Skin         Skin         disease           HUMAN JURKAT         Jurkat Cells         Skin         disease           HUman Colon         Human Colon         Human Colon         Human Colon           Human Thymus         Human Thymus         T-Cell Lymphoma         T-Cell Lymphoma           Lymphoma         Human Adult Heart         Heart         disease           Human Adult Heart         Human Adult Liver,         Human Adult Liver         Human Adult Liver           Human Adult Liver,         Human Whole Six Week Old         Embryo	H0063	Human Thymus	Human Thymus	Thymus			Uni-ZAP XR
Human Activated T-Cells         Blood         Cell Line           Cells         Human Pancreas         Pancreas           Human Infant Adrenal         Human Infant Adrenal Gland         Adrenal gland           Human Infant Adrenal         Human Leiomyeloid         Muscle         disease           Carcinoma         Carcinoma         Activated T-Cells         Blood         Cell Line           Human Activated T-Cells         Blood         Cell Line         disease           Carcinoma         Carcinoma         Cells (II)           Human Activated T-Cells         Blood         Cell Line           Call (II)         Human Thymus Tumor         Thymus           Human Fetal         Human Fetal Skin         Skin           BOUMD         BoUND         Cell Line           Human Fetal         Human Colon         Cell Line           Human Tolon         Human Thymus         Human Thymus           Human Toclil         T-Cell Lymphoma         T-Cell           Human Toclid         Epithelioid Sarcoma, muscle         Sk Muscle           Saccoma         Human Adult Heart         Human Adult Heart           Human Adult Heart         Human Adult Liver         Human Adult Liver           Human Adult Liver         Human Adult Liver	H0068	Human Skin Tumor	Human Skin Tumor	Skin		disease	Uni-ZAP XR
Human Pancreas         Human Pancreas         Pancreas           Human Infant Adrenal         Human Infant Adrenal Gland         Adrenal gland           Gland         Human Leiomyeloid         Muscle         disease           Carcinona         Carcinoma         Carcinoma           Human Activated T-Cells         Activated T-Cells         Blood         Cell Line           Cells (II)         Human Thymus Tumor         Thymus         disease           Human Tetal         Skin         Skin         Adisease           Human Thymus Tumor         Human Fetal Skin         Skin         Adisease           Human Fetal         Jurkat Cells         Skin         Adisease           Human Colon         Human Colon         Human Colon         Human Colon           Human Fibrins         Human Thymus         T-Cell         disease           Human Thymus         Human Thymus         T-Cell         disease           Human Parotid Cancer         Human Adult Heart         Heart         Heart           subtracted         Human Adult Liver         Liver         Liver           subtracted         Human Whole Six         Human Whole Six         Human Whole Six		Human Activated T- Cells	Activated T-Cells	Blood	Cell Line		Uni-ZAP XR
Human Infant Adrenal       Human Infant Adrenal Gland       Adrenal gland         Gland       Human Leiomyeloid       Muscle       disease         Carcinoma       Carcinoma       Carcinoma       disease         Human Activated T-Cells       Blood       Cell Line       disease         Cells (II)       Human Thymus Tumor       Thymus       disease         Human Fetal       Human Fetal       Skin       disease         Bolthelium (Skin)       Jurkat Cells       Skin       disease         HUMAN JURKAT       Jurkat Cells       Skin       disease         BOUND       POLYSOMES       Human Colon       disease         Human Colon       Human Colon       Sk Muscle       disease         Human T-Cell       T-Cell Lymphoma       T-Cell       disease         Human T-Cell       T-Cell Lymphoma       T-Cell       disease         Human Adult Heart       Human Adult Heart       Heart       Heart         Human Adult Liver,       Human Adult Liver       Liver       Liver         subtracted       Human Whole Six Week Old       Embryo	H0070	Human Pancreas	Human Pancreas	Pancreas			Uni-ZAP XR
Human LeiomyeloidHuman LeiomyeloidMusclediseaseCarcinomaCarcinomaCarcinomaGal LineHuman Activated T-CellsBloodCell LineCells (II)Human Thymus TumorThymusHuman Thymus TumorThymusdiseaseHuman Thymus TumorSkindiseaseHuman FetalHuman Fetal SkinSkinBithelium (Skin)Jurkat CellsSkinMEMBRANEHuman ColondiseaseHuman ColonHuman ColondiseaseHuman ColonT-Cell LymphomaT-Cell LymphomaHuman ThymusHuman ThymusT-Cell LymphomaHuman Adult HeartHuman Adult HeartHeartsubtractedHuman Adult LiverLiversubtractedHuman Whole Six Week OldEmbryo	H0071	Human Infant Adrenal Gland	Human Infant Adrenal Gland	Adrenal gland			Uni-ZAP XR
Carcinoma         Carcinoma           Human Activated T-Cells         Blood         Cell Line           Cells (II)         Human Thymus Tumor         Thymus         disease           Human Thymus Tumor         Human Thymus Tumor         Thymus         disease           Human Thymus Tumor         Human Tetal Skin         Skin         disease           HUMAN JURKAT         Jurkat Cells         Skin         disease           BOUND         Human Colon         Human Colon         disease           Human Etal         Epithelioid Sarcoma, muscle         Sk Muscle         disease           Human Colon         Human Thymus         T-Cell Lymphoma         disease           Human Tymus         Human Thymus         T-Cell Lymphoma         disease           Human Adult Heart         Human Adult Heart         Heart         Heart           subtracted         Human Adult Liver         Liver         Liver           subtracted         Human Whole Six Week Old         Embryo         Embryo	H0073	Human Leiomyeloid	Human Leiomyeloid	Muscle		disease	Uni-ZAP XR
Human Activated T-Cells (II)         Activated T-Cells (II)         Blood         Cell Line           Cells (II)         Human Thymus Tumor         Thymus         disease           Human Fetal         Human Fetal Skin         Skin         disease           Epithelium (Skin)         Jurkat Cells         Skin         disease           MEMBRANB         BOUND         POLYSOMES         Human Colon         disease           Human Colon         Human Colon         Sk Muscle         disease           Human petithelioid         Epithelioid Sarcoma, muscle         Sk Muscle         disease           Human T-Cell         T-Cell Lymphoma         T-Cell Lymphoma         disease           Lymphoma         Human Parotid Cancer         Human Adult Heart         Heart           Human Adult Heart,         Human Adult Heart         Heart         Heart           Human Adult Liver,         Human Adult Liver         Liver         Liver           subtracted         Human Whole Six         Human Whole Six Week Old         Embryo		Carcinoma	Carcinoma				
Human Thymus Tumor         Thymus Thymus Tumor         Thymus Disease.           Human Fetal         Human Fetal Skin         Skin         disease.           Human Skin)         Jurkat Cells         American Skin         American Skin           HUMAN JURKAT         Jurkat Cells         American Skin         American Skin           HUMAN JURKAT         Human Colls         American Skin         American Skin           Human Colon         Human Colon         American Skin         American Skin           Human Colon         Human Thymus         T-Cell Lymphoma         American Skin           Human Thymus         T-Cell Lymphoma         American Skin         American Skin           Human Adult Heart         Human Adult Heart         Heart         American Skin           Human Adult Liver         Liver         Liver         Liver           subtracted         Human Whole Six Week Old         Embryo         Embryo	H0075	Human Activated T- Cells (II)	Activated T-Cells	Blood	Cell Line		Uni-ZAP XR
Human Fetal         Human Fetal Skin         Skin           Epithelium (Skin)         Jurkat Cells           HUMAN JURKAT         Jurkat Cells           MEMBRANE         BOUND           POLYSOMES         Human Colon           Human Colon         Human Colon           Human Colon         Epithelioid Sarcoma, muscle           Human Farona         Human Thymus           Human T-Cell         T-Cell Lymphoma           Human T-Cell         T-Cell Lymphoma           Human Parotid Cancer         Parotid           Human Adult Heart         Heart           Human Adult Heart         Heart           Human Adult Liver         Liver           Subtracted         Human Whole Six Week Old           Human Whole Six         Human Whole Six Week Old	H0077	Human Thymus Tumor		Thymus		disease	Lambda ZAP II
Epithelium (Skin)         Jurkat Cells           HUMAN JURKAT         Jurkat Cells           MEMBRANE         Human Colon           Human Colon         Human Colon           Human Colon         Human Thymus           Human Thymus         T-Cell Lymphoma         T-Cell           Human T-Cell         T-Cell Lymphoma         disease           Human Parotid Cancer         Parotid         Heart           Human Adult Heart         Human Adult Liver         Heart           Human Adult Liver         Human Adult Liver         Liver           Human Whole Six Week Old         Embryo	H0081	Human Fetal	Human Fetal Skin	Skin			Uni-ZAP XR
HUMAN JURKAT         Jurkat Cells           MEMBRANE BOUND POLYSOMES         Human Colon         Human Colon           Human colon         Human Colon         disease           Human pithelioid         Epithelioid Sarcoma, muscle         Sk Muscle         disease           Human Thymus         Human Thymus         T-Cell         disease           Human T-Cell         T-Cell Lymphoma         disease           Lymphoma         Human Parotid Cancer         Parotid         disease           Human Adult Heart,         Human Adult Liver         Liver         Liver           Subtracted         Human Whole Six Week Old         Embryo         Embryo		Epithelium (Skin)					
Human Colon Human Colon Human Colon Human Colon Human Colon Human Thymus Human Thymus Human Tr-Cell Lymphoma Human Parotid Cancer Human Adult Heart Human Adult Liver Human Adult Liver Human Adult Liver Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Week Old Human Whole Six Wee	H0083	HUMAN JURKAT	Jurkat Cells				Uni-ZAP XR
POLYSOMESHuman ColonKuman ColonHuman ColonHuman ColonSk MuscleHuman EpithelioidEpithelioid Sarcoma, muscleSk MuscleSarcomaHuman ThymusT-Cell LymphomaHuman T-CellT-Cell LymphomaT-CellLymphomaHuman Parotid CancerParotiddiseaseHuman Adult Heart,Human Adult HeartHeartdiseaseHuman Adult Liver,Human Adult LiverLiverLiversubtractedLiverLiverLiverHuman Whole SixHuman Whole Six Week OldEmbryo		MEMBRANE					
Human ColonHuman ColonSk MusclediseaseSarcoma sarcomaEpithelioid Sarcoma, muscleSk MusclediseaseHuman ThymusT-Cell LymphomaT-CelldiseaseLymphomaTuman Parotid CancerParotiddiseaseHuman Parotid CancerHuman Adult HeartHeartdiseaseHuman Adult Liver,Human Adult LiverLiverLiverSubtractedHuman Adult LiverLiverLiverHuman Whole Six Week OldEmbryoEmbryo		POLYSOMES					
Human epithelioid sarcomaEpithelioid Sarcoma, muscle sarcomaSk MusclediseaseHuman ThymusT-Cell LymphomaT-CellLymphomaT-Cell LymphomadiseaseHuman Parotid CancerParotid Human Adult HeartHeartdiseaseHuman Adult Liver, subtractedHuman Adult LiverLiverLiverHuman Adult Liver, subtractedHuman Adult LiverLiverLiverHuman Whole Six Week OldEmbryoEmbryo	H0085	Human Colon	Human Colon		•		Lambda ZAP II
Human ThymusHuman ThymusT-Cell LymphomaT-CellLymphomaT-Cell LymphomaT-CellLymphomaHuman Parotid CancerParotiddiseaseHuman Adult Heart, subtractedHuman Adult LiverHuman Adult LiverHuman Adult Liver, subtractedHuman Adult LiverLiverSubtractedLiverLiverHuman Whole Six Week OldEmbryo	H0086	Human epithelioid	Epithelioid Sarcoma, muscle	Sk Muscle		disease	Uni-ZAP XR
Human T-CellT-Cell LymphomaT-CelldiseaseLymphomaHuman Parotid CancerParotiddiseaseHuman Adult Heart, subtractedHuman Adult LiverHeartdiseaseHuman Adult Liver, subtractedHuman Adult LiverLiverSubtracted subtractedLiverEmbryo	H0087	Human Thymus	Human Thymus		-		pBluescript
LymphomaHuman Parotid CancerParotiddiseaseHuman Adult HeartHeartHeartSubtractedHuman Adult LiverLiverSubtractedLiverSubtractedEmbryo	H0090	Human T-Cell	T-Cell Lymphoma	T-Cell		disease	Uni-ZAP XR
Human Parotid Cancer         Human Parotid Cancer         Parotid         disease           Human Adult Heart, subtracted         Human Adult Liver         Liver         Liver           Human Adult Liver, subtracted         Human Whole Six Week Old         Embryo		Lymphoma					
Human Adult Heart, Human Adult Heart Heart Subtracted Human Adult Liver Liver Liver Human Whole Six Week Old Embryo	9600H	Human Parotid Cancer	Human Parotid Cancer	Parotid		disease	Lambda ZAP II
Human Adult Liver,Human Adult LiverLiversubtractedEmbryoHuman Whole SixHuman Whole Six Week OldEmbryo	H0097	Human Adult Heart, subtracted	Human Adult Heart	Heart			pBluescript
Human Whole Six Human Whole Six Week Old Embryo	H0098	Human Adult Liver, subtracted	Human Adult Liver	Liver			Uni-ZAP XR
	H0100	Human Whole Six	Human Whole Six Week Old	Embryo			Uni-ZAP XR

Description	Tissue	Organ	Cell Line	Disease	Vector
Week Old Embryo	Embryo				
Human 7 Weeks Old Embrvo, subtracted	Human Whole 7 Week Old Embryo	Embryo			Lambda ZAP II
Human Whole 6 Week Old Embryo (II), subt	Human Whole Six Week Old Embryo	Embryo			pBluescript
Human Fetal Brain, subtracted	. Human Fetal Brain	Brain			Uni-ZAP XR
Human Infant Adrenal Gland, subtracted	Human Infant Adrenal Gland	Adrenal gland			pBluescript
Human Adult Lymph Node, subtracted	Human Adult Lymph Node	Lymph Node			Uni-ZAP XR
Human Old Ovary, subtracted	Human Old Ovary	Ovary			pBluescript
Human Placenta, subtracted	Human Placenta	Placenta			pBluescript
Human Parathyroid Tumor, subtracted	Human Parathyroid Tumor	Parathyroid			pBluescript
Human Thymus Tumor, subtracted	Human Thymus Tumor	Thymus			pBluescript
Human Adult Kidney	Human Adult Kidney	Kidney			Uni-ZAP XR
Human Adult Spleen, subtracted	Human Adult Spleen	Spleen			Uni-ZAP XR
Human Cornea, subtracted	Human Cornea	еуе			Uni-ZAP XR
Human Adult Skeletal Muscle	Human Skeletal Muscle	Sk Muscle			Uni-ZAP XR
Human Fetal Dura Mater	Human Fetal Dura Mater	Brain			Uni-ZAP XR
Human Rhabdomyosarcoma	Human Rhabdomyosarcoma	Sk Muscle		disease	Uni-ZAP XR
Cem cells cyclohexamide treated	Cyclohexamide Treated Cem, Jurkat, Raji, and Supt	Blood	Cell Line		Uni-ZAP XR

Description	Tissue	Organ	Cell Line	Disease	Vector
LNCAP untreated	LNCAP Cell Line	Prostate	Cell Line		Uni-ZAP XR
LNCAP + 0.3nM R1881	LNCAP Cell Line	Prostate	Cell Line		Uni-ZAP XR
LNCAP + 30nM R1881	LNCAP Cell Line	Prostate	Cell Line		Uni-ZAP XR
Raji Cells, cyclohexamide treated	Cyclohexamide Treated Cem, Jurkat, Raji, and Supt	Blood	Cell Line		Uni-ZAP XR
Human Synovial Sarcoma	Human Synovial Sarcoma	Synovium			Uni-ZAP XR
Supt Cells, cyclohexamide treated	Cyclohexamide Treated Cem, Jurkat, Raji, and Supt	Blood	Cell Line		Uni-ZAP XR
Activated T-Cells, 4 hrs.	Activated T-Cells	Blood	Cell Line		Uni-ZAP XR
Activated T-Cells, 8 hrs.	Activated T-Cells	Blood	Cell Line		Uni-ZAP XR
Activated T-Cells, 12 hrs.	Activated T-Cells	Blood	Cell Line		Uni-ZAP XR
Nine Week Old Early Stage Human	9 Wk Old Early Stage Human	Embryo			Uni-ZAP XR
Human Adult Liver	Human Adult Liver	Liver			Uni-ZAP XR
7 Week Old Early Stage Human,	Human Whole 7 Week Old Embryo	Embryo			Uni-ZAP XR
Human Epididymus	Epididymis	Testis	-		Uni-ZAP XR
Early Stage Human Liver	Human Fetal Liver	Liver			Uni-ZAP XR
Human Fibrosarcoma	Human Skin Fibrosarcoma	Skin		disease	Uni-ZAP XR
Human Adrenal Gland Tumor	Human Adrenal Gland Tumor	Adrenal Gland		disease	Uni-ZAP XR
Activated T-Cells, 4 hrs., ligation 2	Activated T-Cells	Blood	Cell Line		Uni-ZAP XR
Activated T-Cells, 8	Activated T-Cells	Blood	Cell Line		Uni-ZAP XR

		JP XR	JP XR	IP XR	AP XR	AP XR	∿ XR	AP XR	AP XR	JP XR	Lambda ZAP II	₽ XR	VP XR	\P XR	LP XR	\P XR	LP XR	
Vector		Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Lambd	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	/Z-iuO	Uni-ZAP XR	_
Disease					disease	disease		disease								disease	disease	
Cell Line		Cell Line	Cell Line				Cell Line					Cell Line	Cell Line		Cell Line			
Organ		Blood	Blood	Synovium	Prostate	Prostate	Blood	Prostate	Embryo	Embryo	Brain	Breast	Breast	Brain	Blood	Breast	Breast	
Tissue		Activated T-Cells	Activated T-Cells	Human Synovium	Human Prostate Cancer, stage B2	Human Prostate Cancer, stage B2	Activated T-Cells	Human Prostate Cancer, stage C	Twelve Week Old Early Stage Human	Twelve Week Old Early Stage Human	Human Fetal Brain	CAMA1Ee Cell Line	CAMA1Ee Cell Line	Human Fetal Brain	Human Neutrophil	Human Primary Breast Cancer	Human Primary Breast	Californ
Description	hrs., ligation 2	Activated T-Cells, 12 hrs., ligation 2	Activated T-Cells, 24 hrs., ligation 2	Human Synovium	Human Prostate Cancer, Stage B2	Human Prostate Cancer, Stage B2 fraction	Activated T-Cells, 24 hrs.	Human Prostate Cancer, Stage C fraction	12 Week Old Early Stage Human	12 Week Old Early Stage Human, II	Human Fetal Brain, random primed	CAMA I Ee Cell Line	CAMA1Ee Cell Line	Human Fetal Brain	Human Neutrophil	Human Primary Breast Cancer	Human Primary Breast	Callet
Code		H0160	H0161	H0163	H0165	H0166	H0167	H0169	H0170	H0171	H0172	H0176	H0177	H0178	H0179	H0180	H0181	-

Tissue Human Colon Cancer	Ö	Organ	Cell Line	Disease disease	Vector Uni-ZAP XR
T-Cells		Blood	Cell Line	OGBOGIE.	Lambda ZAP II
Human Normal Breast		Breast			Uni-ZAP XR
Human Macrophage/Monocytes		Blood	Cell Line		Uni-ZAP XR
Cyclohexamide Treated Cem, Jurkat, Raji, and Supt	i	Blood	Cell Line		Uni-ZAP XR
Human Cerebellum		Brain			pBluescript
Human Cardiomyopathy		Heart			Uni-ZAP XR
Human Fetal Liver		Liver			Uni-ZAP XR
Human Fetal Liver		Liver			Uni-ZAP XR
Human Greater Omentum pe	<del> </del>	peritoneum			Uni-ZAP XR
Human Hippocampus		Brain			pBluescript
Human Colon Cancer		Colon	-		pBluescript
Human Colon Cancer		Colon			pBluescript
LNCAP Cell Line		Prostate	Cell Line		pBluescript
Human Fetal Lung		Lung			pBluescript
Human Cerebellum		Brain			Uni-ZAP XR

$\Box$	Tissue Organ Human Prostate Pro	an Prostate	Cell Line	Disease	Vector pBluescript
Human Prostate		Prostate			pBluescript
Human Pituitary	itary				Uni-ZAP XR
Cyclohexamide Treated Cem, Jurkat, Raji, and Supt		Blood	Cell Line		pBluescript
Cyclohexamide Treated C Jurkat, Raji, and Supt	em,	Blood	Cell Line		pBluescript
Cyclohexamide Treated C Jurkat, Raji, and Supt	em,	Blood	Cell Line		pBluescript
Activated T-Cells		Blood	Cell Line		Uni-ZAP XR
Activated T-Cells		Blood	Cell Line		Uni-ZAP XR
Activated T-Cells		Blood	Cell Line		Uni-ZAP XR
Activated T-Cells		Blood	Cell Line		Uni-ZAP XR
Activated T-Cells		Blood	Cell Line		Uni-ZAP XR
Activated T-Cells		Blood	Cell Line		Uni-ZAP XR
Human Cardio	Cardiomyopathy	Heart		disease	Uni-ZAP XR

	Description	Tissue	Organ	Cell Line	Disease	Vector
,						
la s	Human Colon, subtraction	Human Colon				pBluescript
nar	Human Colon, differential expression	Human Colon				pBluescript
Human F Different Specific)	Human Fetal Heart, Differential (Adult- Specific)	Human Fetal Heart	Heart	·		pBluescript
tatic	Human colon cancer, metaticized to liver, subtraction	Human Colon Cancer, metasticized to liver	Liver			pBluescript
ımar	Human Kidney Tumor	Human Kidney Tumor	Kidney		disease	Uni-ZAP XR
Human F Different Specific)	Human Fetal Heart, Differential (Fetal- Specific)	Human Fetal Heart	Неап			pBluescript
umai mbry	Human 8 Week Whole Embryo, subtracted	Human 8 Week Old Embryo	Embryo			Uni-ZAP XR
umai	Human Fetal Liver- Enzyme subtraction	Human Fetal Liver	Liver			Uni-ZAP XR
Iono	Human Activated Monocytes	Human Monocytes				Uni-ZAP XR
Human Chondr	Human Chondrosarcoma	Human Chondrosarcoma	Cartilage		disease	Uni-ZAP XR
umai	Human Osteosarcoma	Human Osteosarcoma	Вопе		disease	Uni-ZAP XR
umai rge i	Human adult testis, large inserts	Human Adult Testis	Testis			Uni-ZAP XR
reast	Breast Lymph node cDNA library	Breast Lymph Node	Lymph Node			Uni-ZAP XR
east	breast lymph node CDNA library	Breast Lymph Node	Lymph Node			Lambda ZAP II
T-60	HL-60, unstimulated	Human HL-60 Cells, unstimulated	Blood	Cell Line		Uni-ZAP XR

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0257	HL-60, PMA 4H	HL-60 Cells, PMA stimulated 4H	Blood	Cell Line		Uni-ZAP XR
H0261	H. cerebellum, Enzyme subtracted	Human Cerebellum	Brain			Uni-ZAP XR
H0263	human colon cancer	Human Colon Cancer	Colon		disease	Lambda ZAP II
H0264	human tonsils	Human Tonsil	Tonsil			Uni-ZAP XR
H0265	Activated T-Cell (12hs)/Thiouridine labelledEco	T-Cells	Blood	Cell Line		Uni-ZAP XR
Н0266	Human Microvascular Endothelial Cells, fract. A	HMEC	Vein	Cell Line		Lambda ZAP II
H0267	Human Microvascular Endothelial Cells, fract. B	HMEC	Vein	Cell Line		Lambda ZAP II
H0268	Human Umbilical Vein Endothelial Cells, fract. A	HUVE Cells	Umbilical vein	Cell Line		Lambda ZAP II
H0269	Human Umbilical Vein Endothelial Cells, fract. B	HUVE Cells	Umbilical vein	Cell Line		Lambda ZAP II
H0270	HPAS (human pancreas, subtracted)	Human Pancreas	Pancreas			Uni-ZAP XR
H0271	Human Neutrophil, Activated	Human Neutrophil - Activated	Blood	Cell Line		Uni-ZAP XR
H0272	HUMAN TONSILS, FRACTION 2	Human Tonsil	Tonsil			Uni-ZAP XR
H0274	Human Adult Spleen, fractionII	Human Adult Spleen	Spleen			Uni-ZAP XR
H0275	Human Infant Adrenal Gland, Subtracted	Human Infant Adrenal Gland	Adrenal gland			pBluescript
H0280	K562 + PMA (36 hrs)	K562 Cell line	cell line	Cell Line		ZAP Express

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0282	HBGB"s differential	man	Breast			Uni-ZAP XR
	consolidation	Cancer				
H0284	Human OB MG63	Human Osteoblastoma MG63 cell line	Bone	Cell Line		Uni-ZAP XR
H0286	Human OB MG63	Human Osteoblastoma MG63	Bone	Cell Line		Uni-ZAP XR
	treated (10 nM E2)	cell line				
H0288	Human OB HOS	Human Osteoblastoma HOS	Bone	Cell Line		Uni-ZAP XR
	control fraction I	cell line				
H0290	Human OB HOS	Human Osteoblastoma HOS	Bone	Cell Line		Uni-ZAP XR
	treated (1 nM E2) fraction I	cell line				
H0292	Human OB HOS	Human Osteoblastoma HOS	Bone	Cell Line		Uni-ZAP XR
	treated (10 nM E2)	cell line				
	rraction 1					
H0294	Amniotic Cells - TNF induced	Amniotic Cells - TNF induced	Placenta	Cell Line		Uni-ZAP XR
H0295	Amniotic Cells -	Amniotic Cells - Primary	Placenta	Cell Line	-	Uni-ZAP XR
	Primary Culture	Culture				
H0298	HCBB's differential consolidation	CAMA1Ee Cell Line	Breast	Cell Line		Uni-ZAP XR
H0305	CD34 positive cells (Cord Blood)	CD34 Positive Cells	Cord Blood			ZAP Express
H0306	CD34 depleted Buffy Coat (Cord Blood)	CD34 Depleted Buffy Coat (Cord Blood)	Cord Blood	-		ZAP Express
H0309	Human Chronic Synovitis	Synovium, Chronic Synovitis/ Osteoarthritis	Synovium		disease	Uni-ZAP XR
H0310	human caudate nucleus	Brain	Brain			Uni-ZAP XR
H0313	human pleural cancer	pleural cancer			disease	pBluescript
H0316	HUMAN STOMACH	Human Stomach	Stomach			Uni-ZAP XR
H0318	HUMAN B CELL LYMPHOMA	Human B Cell Lymphoma	Lymph Node		disease	Uni-ZAP XR

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0320	Human frontal cortex	Human Frontal Cortex	Brain			Uni-ZAP XR
H0321	HUMAN SCHWANOMA	Schwanoma	Nerve		disease	Uni-ZAP XR
H0327	human corpus colosum	Human Corpus Callosum	Brain			Uni-ZAP XR
H0328	human ovarian cancer	Ovarian Cancer	Ovary		disease	Uni-ZAP XR
H0329	Dermatofibrosarcoma Protuberance	Dermatofibrosarcoma Protuberans	Skin		disease	Uni-ZAP XR
H0331	Hepatocellular Tumor	Hepatocellular Tumor	Liver		disease	Lambda ZAP II
H0333	Hemangiopericytoma	Hemangiopericytoma	Blood vessel		disease	Lambda ZAP II
H0334	Kidney cancer	Kidney Cancer	Kidney		disease	Uni-ZAP XR
H0339	Duodenum	Duodenum				Uni-ZAP XR
H0341	Bone Marrow Cell Line (RS4:11)	Bone Marrow Cell Line RS4;11	Bone Marrow	Cell Line		Uni-ZAP XR
H0343	stomach cancer (human)	Stomach Cancer - 5383A (human)			disease	Uni-ZAP XR
H0345	SKIN	Skin - 4000868H	Skin			Uni-ZAP XR
H0346	Brain-medulloblastoma	Brain (Medulloblastoma)- 9405C006R	Brain		disease	Uni-ZAP XR
H0349	human adult liver cDNA library	Human Adult Liver	Liver			pCMVSport 1
H0350	Human Fetal Liver, mixed 10 & 14 week	Human Fetal Liver, mixed 10&14 Week	Liver			Uni-ZAP XR
H0351	Glioblastoma	Glioblastoma	Brain		disease	Uni-ZAP XR
H0352	wilm"s tumor	Wilm's Tumor			disease	Uni-ZAP XR
H0354	Human Leukocytes	Human Leukocytes	Blood	Cell Line		pCMVSport 1
H0355	Human Liver	Human Liver, normal Adult				pCMVSport 1
H0356	Human Kidney	Human Kidney	Kidney			pCMVSport 1
H0357	H. Normalized Fetal	Human Fetal Liver	Liver			Uni-ZAP XR
H0359	KMH2 cell line	KMH2				ZAP Express
H0361	Human rejected kidney	Human Rejected Kidney			disease	pBluescript

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0362	HeLa cell line	HELA CELL LINE				pSport1
H0366	L428 cell line	L428				ZAP Express
H0369	H. Atrophic Endometrium	Atrophic Endometrium and myometrium				Uni-ZAP XR
H0370	H. Lymph node breast Cancer	Lymph node with Met. Breast Cancer			disease	Uni-ZAP XR
H0372	Human Testes	Human Testes	Testis			pCMVSport 1
H0373	Human Heart	Human Adult Heart	Heart			pCMVSport 1
H0374	Human Brain	Human Brain				pCMVSport 1
H0375	Human Lung	Human Lung				pCMVSport 1
H0376	Human Spleen	Human Adult Spleen	Spleen			pCMVSport 1
H0379	Human Tongue, frac 1	Human Tongue				pSport1
H0380	Human Tongue, frac 2	Human Tongue				pSport1
H0381	Bone Cancer	Bone Cancer			disease	Uni-ZAP XR
H0383	Human Prostate BPH,	Human Prostate BPH				Uni-ZAP XR
	re-excision					
H0384	Brain, Kozak	Human Brain				pCMVSport 1
H0386	Leukocyte and Lung; 4 screens	Human Leukocytes	Blood	Cell Line		pCMVSport 1
H0388	Human Rejected Kidney, 704 re- excision	Human Rejected Kidney			disease	pBluescript
H0390	Human Amygdala Depression, re-excision	Human Amygdala Depression			disease	pBluescript
H0391	H. Meniingima, M6	Human Meningima	brain			pSport1
H0392	H. Meningima, M1	Human Meningima	brain			pSport1
H0393	Fetal Liver, subtraction II	Human Fetal Liver	Liver			pBluescript
H0394	A-14 cell line	Redd-Sternberg cell				ZAP Express
H0395	A1-CELL LINE	Redd-Sternberg cell				ZAP Express
H0399	Human Kidney Cortex, re-rescue	Human Kidney Cortex				Lambda ZAP II

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0400	Human Striatum	Human Brain, Striatum	Brain			Lambda ZAP II
	Depression, re-rescue	Depression				
H0402	CD34 depleted Buffy Coat (Cord Blood), re-	CD34 Depleted Buffy Coat (Cord Blood)	Cord Blood			ZAP Express
H0403	H. Umbilical Vein Endothelial Cells, IL4	HUVE Cells	Umbilical vein	Cell Line		Uni-ZAP XR
	induced					
H0404	H. Umbilical Vein	HUVE Cells	Umbilical vein	Cell Line		Uni-ZAP XR
	endothelial cells, uninduced					
H0405	Human Pituitary,	Human Pituitary				pBluescript
	subtracted VI					
H0406	H Amygdala Depression, subtracted	Human Amygdala Depression				Uni-ZAP XR
H0408	Human kidney Cortex, subtracted	Human Kidney Cortex				pBluescript
H0409	H. Striatum	Human Brain, Striatum	Brain			pBluescript
	Depression, subtracted	Depression				
H0410	H. Male bladder, adult	H Male Bladder, Adult	Bladder			pSport1
H0411	H Female Bladder, Adult	Human Female Adult Bladder	Bladder			pSport1
H0412	Human umbilical vein	HUVE Cells	Umbilical vein	Cell Line		pSport1
	endothelial cells, IL-4 induced			-		
H0413	Human Umbilical Vein	HUVE Cells	Umbilical vein	Cell Line		pSport1
	Endothelial Cells, uninduced					
H0415	H. Ovarian Tumor, П, OV5232	Ovarian Tumor, OV5232	Ovary	•	disease	pCMVSport 2.0
H0416	Human Neutrophils, Activated, re-excision	Human Neutrophil - Activated	Blood	Cell Line		pBluescript

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0417	Human Pituitary,	Human Pituitary				pBluescript
H0418	Human Pituitary, subtracted VII	Human Pituitary				pBluescript
H0419	Bone Cancer, re- excision	Bone Cancer				Uni-ZAP XR
H0421	Human Bone Marrow, re-excision	Bone Marrow				pBluescript
H0422	T-Cell PHA 16 hrs	T-Cells	Blood	Cell Line		pSport1
H0423	T-Cell PHA 24 hrs	T-Cells	Blood	Cell Line		pSport1
H0424		Human Pituitary				pBluescript
H0427	Human Adipose	Human Adipose, left hiplipoma	3	-		pSport1
H0428	Human Ovary	Human Ovary Tumor	Ovary			pSport1
H0429	K562 + PMA (36 hrs),re-excision	K562 Cell line	cell line	Cell Line		ZAP Express
H0431	H. Kidney Medulla, re- excision	Kidney medulla	Kidney			pBluescript
H0433	Human Umbilical Vein Endothelial cells, frac B, re-excision	HUVE Cells	Umbilical vein	Cell Line		pBluescript
H0435	Ovarian Tumor 10-3- 95	Ovarian Tumor, OV350721	Ovary			pCMVSport 2.0
H0436	Resting T-Cell Library,II	T-Cells	Blood	Cell Line		pSport1
H0437	H Umbilical Vein Endothelial Cells, frac A, re-excision	HUVE Cells	Umbilical vein	Cell Line		Lambda ZAP II
H0438	H. Whole Brain #2, re- excision	Human Whole Brain #2				ZAP Express
H0439	₩	Eosinophils				pBluescript

Code	Description	Tissua	Organ	Coll I ino	Dicooca	Vertor
H0441	H. Kidney Cortex,	Kidney cortex	Kidney			pBluescript
	subtracted		•			•
H0443	H. Adipose, subtracted	Human Adipose, left hiplipoma				pSport1
H0444	Spleen metastic melanoma	Spleen, Metastic malignant melanoma	Spleen	·	disease	pSport1
H0445	Spleen, Chronic lymphocytic leukemia	Human Spleen, CLL	Spleen		disease	pSport1
H0449	CD34+ cell, I	CD34 positive cells				pSport1
H0455	H. Striatum	Human Brain, Striatum	Brain			pBluescript
H0457	Depression, subt Himan Fosinophils	Depression Human Fosinophile				nSport 1
H0458	CD34+ cell, I, frac II	CD34 positive cells				pSport1
H0459	CD34+cells, II, FRACTION 2	CD34 positive cells				pCMVSport 2.0
H0461	H. Kidney Medulla, subtracted	Kidney medulla	Kidney			pBluescript
H0477	Human Tonsil, Lib 3	Human Tonsil	Tonsil			pSport1
H0478	Salivary Gland, Lib 2	Human Salivary Gland	Salivary gland			pSport1
H0479	Salivary Gland, Lib 3	Human Salivary Gland	Salivary gland			pSport1
H0483	Breast Cancer cell line, MDA 36	Breast Cancer Cell line, MDA 36				pSport1
H0484	Breast Cancer Cell line, angiogenic	Breast Cancer Cell line, Angiogenic, 36T3		·		pSport1
H0485	Hodgkin"s Lymphoma I	Hodgkin"s Lymphoma I			disease	pCMVSport 2.0
H0486	Hodgkin"s Lymphoma II	Hodgkin"s Lymphoma II			disease	pCMVSport 2.0
H0487	Human Tonsils, lib I	Human Tonsils				pCMVSport 2.0
H0488	Human Tonsils, Lib 2	Human Tonsils				pCMVSport 2.0
H0489	Crohn"s Disease	Ileum	Intestine		disease	pSport1
H0491	HL-60, PMA 4H,	HL-60 Cells, PMA stimulated	Blood	Cell Line		Uni-ZAP XR

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
	subtracted	4H				
H0492	HL-60, RA 4h, Subtracted	HL-60 Cells, RA stimulated for 4H	Blood	Cell Line		Uni-ZAP XR
H0494	Keratinocyte	Keratinocyte				pCMVSport 2.0
H0497	HEL cell line	HEL cell line		HEL 92.1.7		pSport 1
H0505	Human Astrocyte	Human Astrocyte				pSport1
H0506		Colon	Colon			pSport1
H0509	Liver, Hepatoma	Human Liver, Hepatoma, patient 8	Liver		disease	pCMVSport 3.0
H0510	Human Liver, normal	Human Liver, normal, Patient #8	Liver			pCMVSport 3.0
H0512	Keratinocyte, lib 3	Keratinocyte		•		pCMVSport 2.0
H0518	pBMC stimulated w/	pBMC stimulated with poly I/C				pCMVSport 3.0
H0519	NTERA2, control	NTERA2, Teratocarcinoma cell line				pCMVSport 3.0
H0520	NTERA2 + retinoic acid, 14 days	NTERA2, Teratocarcinoma cell line				pSport1
H0521	Primary Dendritic Cells, lib 1	Primary Dendritic cells				pCMVSport 3.0
H0522	Primary Dendritic cells, frac 2	Primary Dendritic cells				pCMVSport 3.0
H0525	PCR, pBMC I/C treated	pBMC stimulated with poly I/C		٠		PCRII
H0529	Myoloid Progenitor Cell Line	TF-1 Cell Line; Myoloid progenitor cell line				pCMVSport 3.0
H0530	Human Dermal Endothelial Cells,untreated	Human Dermal Endothelial Cells; untreated				pSport1
H0537	H. Primary Dendritic Cells,lib 3	Primary Dendritic cells				pCMVSport 2.0
H0538	Merkel Cells	Merkel cells	Lymph node			pSport1

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
┼─		Pancreas Islet Cell Tumour	Pancreas		disease	pSport1
H0542	T Cell helper I	Helper T cell				pCMVSport 3.0
╁	T cell helper II	Helper T cell				pCMVSport 3.0
+	Human endometrial	Human endometrial stromal				pCMVSport 3.0
	stromal cells	cells				
H0545	Human endometrial	Human endometrial stromal				pCMVSport 3.0
	stromal cells-treated	cells-treated with proge				
-	WILLI DIOBESICIONE	1				nCMVSnort 3.0
H0546	Human endometrial	Human endometrial stromal				perm v sport 3.0
	with estradiol	נכווז-עיכמונע איווו נאדמ				
H0547	NTERA2	NTERA2, Teratocarcinoma				pSport1
	teratocarcinoma cell	cell line				
	line+retinoic acid (14					_
	days)					
H0549	H. Epididiymus, caput	Human Epididiymus, caput				Uni-ZAP XR
	& corpus	and corpus				
H0550	H. Epididiymus, cauda	Human Epididiymus, cauda				Uni-ZAP XR
H0551	Human Thymus	Human Thymus Stromal				pCMVSport 3.0
┥	Stromal Cells	Cells				0.67 - 0.63 40
H0553	Human Placenta	Human Placenta				pCM v Sport 3.0
H0555	Rejected Kidney, lib 4	Human Rejected Kidney	Kidney		disease	pCMVSport 3.0
H0556	Activated T-	T-Cells	Blood	Cell Line		Uni-ZAP XR
	cell(12h)/Thiouridine-					
	re-excision					
H0559	HL-60, PMA 4H, re-	HL-60 Cells, PMA stimulated	Blood	Cell Line		Uni-ZAP XR
	excision	4H				
H0560	KMH2	KMH2				pCMVSport 3.0
H0561	L428	L428				pCMVSport 3.0
Н0562	Human Fetal Brain, normalized c5-11-26	Human Fetal Brain	,			pCMVSport 2.0

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0563	Human Fetal Brain, normalized 50021F	Human Fetal Brain				pCMVSport 2.0
H0564	Human Fetal Brain, normalized C5001F	Human Fetal Brain				pCMVSport 2.0
H0566	Human Fetal Brain,normalized c50F	Human Fetal Brain				pCMVSport 2.0
H0567	Human Fetal Brain, normalized A5002F	Human Fetal Brain				pCMVSport 2.0
H0569	Human Fetal Brain, normalized CO	Human Fetal Brain				pCMVSport 2.0
H0570	Human Fetal Brain, normalized C500H	Human Fetal Brain				pCMVSport 2.0
H0571	Human Fetal Brain, normalized C500HE	Human Fetal Brain				pCMVSport 2.0
H0572	Human Fetal Brain, normalized AC5002	Human Fetal Brain				pCMVSport 2.0
H0574	Hepatocellular Tumor; re-excision	Hepatocellular Tumor	Liver		disease	Lambda ZAP II
H0575	Human Adult Pulmonary;re-excision	Human Adult Pulmonary	Lung			Uni-ZAP XR
H0576	Resting T-Cell; re- excision	T-Cells	Blood	Cell Line		Lambda ZAP II
H0578	Human Fetal Thymus	Fetal Thymus	Thymus			pSport1
H0580	Dendritic cells, pooled	Pooled dendritic cells			,	pCMVSport 3.0
H0581	Human Bone Marrow, treated	Human Bone Marrow	Bone Marrow			pCMVSport 3.0
H0583	B Cell lymphoma	B Cell Lymphoma	B Cell		disease	pCMVSport 3.0
H0584	Activated T-cells, 24 hrs,re-excision	Activated T-Cells	Blood	Cell Line	-	Uni-ZAP XR
H0585	Activated T-Cells,12 hrs,re-excision	Activated T-Cells	Blood	Cell Line		Uni-ZAP XR
H0586	Healing groin wound,	healing groin wound, 6.5	groin		disease	pCMVSport 3.0

Vector		pCMVSport 3.0	ZAP Express	Uni-ZAP XR	Uni-ZAP XR	pCMVSport 3.0		pCMVSport 3.0	Lambda ZAP II	Uni-ZAP XR		Lambda ZAP 11	Lambda ZAP II Lambda ZAP II	ambda ZAP II ambda ZAP II	Lambda ZAP II Lambda ZAP II Uni-ZAP XR	Lambda ZAP II Lambda ZAP II Uni-ZAP XR Uni-ZAP XR	Lambda ZAP 11  Lambda ZAP II  Uni-ZAP XR  Uni-ZAP XR  pCMVSport 3.0
Disease Ve		disease pC	ZA	Ū.	disease Un	disease pC		pC	disease La	disease Un		<u> </u>				CI CI Fa	La La Ur disease pC
Cell Line Dise																	
Organ Ce		groin	Cord Blood	Small Int.	T-Cell				Lung			· Colon	Colon	Colon	. Colon Stomach	Colon Stomach Heart	Colon Stomach Heart
Tissue	rs post incision - 2/	Groin-2/19/97	CD34 Positive Cells	Human Adult Small Intestine	T-Cell Lymphoma	HGS wound healing project;	abdomen	Olfactory epithelium from roof of left nasal cacit	Human Lung Cancer	Stomach Cancer - 5383A		Human Colon Cancer	Human Colon Cancer Human Colon	Human Colon Cancer Human Colon	Human Colon Cancer Human Colon Human Stomach	Human Colon Human Colon Human Stomach Human Adult Heart	Human Colon Human Stomach Human Adult Heart Abdomen
Description	sion	Healing groin wound; 7.5 hours post incision	CD34 positive cells (cord blood),re-ex	Human adult small intestine,re-excision	Human T-cell	Healing groin wound -	zero hr post-incision (control)	Olfactory epithelium;nasalcavity	Human Lung Cancer:re-excision	Stomach cancer	(manual), cocomo	Human Colon	Human Colon Cancer;re-excision Human Colon; re-	Human Colon Cancer;re-excision Human Colon; re-	Human Colon Cancer,re-excision Human Colon; re- excision Human Stomach;re- excision	Human Colon Cancer;re-excision Human Colon; re- excision Human Stomach;re- excision Human Adult Heart;re- excision	Human Colon Cancer;re-excision Human Colon; re- excision Human Stomach;re- excision Human Adult Heart;re- excision Human Adult Heart;re- excision Healing Abdomen wound;70&90 min
Code	一	H0587 F	H0589 (	H0590	H0591	H0592 1		H0593 (	H0594 I	H0595	H0596	_				<del></del>	

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0602	Healing Abdomen	Abdomen			disease	pCMVSport 3.0
	Wound;21&29 days					
	post incision					aD luccosint
H0604	Human Pituitary, re- excision	Human Pituitary				poluescripi
9090H	Human Primary Breast	Human Primary Breast	Breast		disease	Uni-ZAP XR
	Cancer;re-excision	Cancer				1. 0.00
H0611	H. Leukocytes,	H.Leukocytes				pCMVSport 1
	normalized cot 500 B					
H0613	H.Leukocytes,	H.Leukocytes				pCMVSport 1
	normalized cot 5B					
H0614	H. Leukocytes,	H.Leukocytes				pCMVSport 1
-	normalized cot 500 A					
H0615	Human Ovarian	Ovarian Cancer	Ovary		disease	Uni-ZAP XR
	Cancer Reexcision					
H0616	Human Testes,	Human Testes	Testis			Uni-ZAP XR
	Reexcision					0/2 d / 2 / 11
H0617	Human Primary Breast	Human Primary Breast	Breast		disease	Uni-ZAP XK
	Cancer Reexcision	Cancer				
H0618	Human Adult Testes,	Human Adult Testis	Testis			Uni-ZAP XR
	Large Inserts,					
H0619	Fetal Heart	Human Fetal Heart	Heart			Uni-ZAP XR
H0620	Human Fetal Kidney;	Human Fetal Kidney	Kidney		-	Uni-ZAP XR
	Reexcision					
H0622	Human Pancreas	Human Pancreas Tumor	Pancreas		disease	Uni-ZAP XR
	Tumor; Reexcision					
H0623	Human Umbilical	Human Umbilical Vein	Umbilical vein			Uni-ZAP XR
	Vein; Reexcision	Endothelial Cells				
H0624	12 Week Early Stage	Twelve Week Old Early	Embryo		<del></del>	Uni-ZAP XK
	-	Stage Liuitan				202041
H0625	Ku 812F Basophils	Ku 812F Basophils				pspont

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
	Line					
H0626	Saos2 Cells; Untreated	Saos2 Cell Line; Untreated				pSport1
H0627	Saos2 Cells; Vitamin D3 Treated	Saos2 Cell Line; Vitamin D3 Treated				pSport1
H0628	Human Pre-	Human Pre-Differentiated				Uni-ZAP XR
	Differentiated Adipocytes	Adipocytes				
H0631	Saos2,	Saos2 Cell Line;				pSport1
	Dexamethosome Treated	Dexamethosome Treated				
H0632	Hepatocellular Tumor:re-excision	Hepatocellular Tumor	Liver			Lambda ZAP II
H0633	Lung Carcinoma A549 TNFaloha activated	TNFalpha activated A549 Lung Carcinoma			disease	pSport1
H0634	Human Testes Tumor,	Human Testes Tumor	Testis		disease	Uni-ZAP XR
H0635	Human Activated T- Cells. re-excision	Activated T-Cells	Blood	Cell Line		Uni-ZAP XR
H0637	Dendritic Cells From CD34 Cells	Dentritic cells from CD34 cells				pSport1
H0638	CD40 activated monocyte dendridic cells	CD40 activated monocyte dendridic cells				pSportl
H0640	Ficolled Human Stromal Cells, Untreated	Ficolled Human Stromal Cells, Untreated				Other
H0641	LPS activated derived dendritic cells	LPS activated monocyte derived dendritic cells				pSport1
H0642	Hep G2 Cells, lambda library	Hep G2 Cells				Other
H0643	Hep G2 Cells, PCR library	Hep G2 Cells				Other

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0644	Human Placenta (re-	Human Placenta	Placenta			Uni-ZAP XR
	excision)					
H0645	Fetal Heart, re-excision	Human Fetal Heart	Heart			Uni-ZAP XR
H0646	Lung, Cancer	Metastatic squamous cell			<u></u>	pSport1
	(4005313 A3):	lung carcinoma, poorly di				
	Invasive Poorly					
	Differentiated Lung					
	Adenocarcinoma,				•	
H0647	Lung, Cancer	Invasive poorly differentiated			disease	pSportl
	(4005163 B7):	lung adenocarcinoma				
	Invasive, Poorly Diff.					
_	Adenocarcinoma,					
	Metastatic					
H0648	Ovary, Cancer:	Papillary Cstic neoplasm of			disease	pSport1
	(4004562 B6)					
	Papillary Serous Cystic					
	Neoplasm, Low					
	Malignant Pot					
H0649	Lung, Normal: (4005313 B1)	Normal Lung				pSport1
H0650	B-Cells	B-Cells				pCMVSport 3.0
H0651	Ovary, Normal:	Normal Ovary				pSport1
	-					
H0652	Lung, Normal: (4005313 B1)	Normal Lung		-		pSport1
H0653	Stromal Cells	Stromal Cells				pSport1
H0656	╁	B-cells (unstimulated)				pSport1
H0657	B-cells (stimulated)	B-cells (stimulated)				pSport1
H0658	Ovary, Cancer	9809C332- Poorly	Ovary &		disease	pSport1
	(9809C332): Poorly	differentiate	Fallopian Tubes			
	differentiated					
	adenocarcinoma					

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0659	Ovary, Cancer	Grade II Papillary Carcinoma,	Ovary		disease	pSport1
	(15395A1F): Grade II Papillary Carcinoma	Ovary				
0990Н	Ovary, Cancer: (15799A1F) Poorly	Poorly differentiated carcinoma, ovary			disease	pSponl
	differentiated carcinoma					
H0661	Breast, Cancer: (4004943 A5)	Breast cancer			disease	pSporti
H0662	Breast, Normal: (4005522B2)	Normal Breast - #4005522(B2)	Breast			pSport1
H0663	Breast, Cancer: (4005522 A2)	Breast Cancer - #4005522(A2)	Breast		disease	pSport1
H0664	Breast, Cancer: (9806C012R)	Breast Cancer	Breast		disease	pSport1
H0665	Stromal cells 3.88	Stromal cells 3.88				pSport1
9990H		Ovarian Cancer, Sample #4004332A2			disease	pSport1
Н0667	Stromal cells(HBM3.18)	Stromal cell(HBM 3.18)				pSportl
H0668	stromal cell clone 2.5	stromal cell clone 2.5	=			pSport1
6990H	Breast, Cancer: (4005385 A2)	Breast Cancer (4005385A2)	Breast			pSport1
H0670	Ovary,	Ovarian Cancer - 4004650A3				pSport1
	Well-Differentiated Micropanillary Sergie	·				
	Carcinoma					
H0671	Breast, Cancer: (9802C02OE)	Breast Cancer- Sample # 9802C02OE				pSport1
H0672	Ovary, Cancer: (4004576 A8)	Ovarian Cancer(4004576A8)	Ovary			pSport1

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
H0673	Human Prostate	Prost	Prostate			Uni-ZAP XR
	Cancer, Stage B2; re- excision	B2				
H0674	Human Prostate Cancer, Stage C; re-	Human Prostate Cancer, stage C	Prostate			Uni-ZAP XR
	excission					
H0675	Colon, Cancer: (9808C064R)	Colon Cancer 9808C064R				pCMVSport 3.0
H0677	TNFR degenerate oligo	B-Cells				PCRII
H0678	screened clones from placental library	Placenta	Placenta			Other
H0682	Serous Papillary Adenocarcinoma	serous papillary adenocarcinoma				pCMVSport 3.0
		(9606G304SPA3B)				
H0683	Ovarian Serous	Serous papillary				pCMVSport 3.0
	Papillary	adenocarcinoma, stage 3C				
	Adenocarcinoma	(9804G01				
H0684	Serous Papillary Adenocarcinoma	Ovarian Cancer-9810G606	Ovaries			pCMVSport 3.0
H0685	Adenocarcinoma of	Adenocarcinoma of Ovary,				pCMVSport 3.0
	Ovary, Human Cell Line, # OVCAR-3	Human Cell Line, # OVCAR-				
H0686	Adenocarcinoma of	Adenocarcinoma of Ovary,				pCMVSport 3.0
	Ovary, Human Cell	Human Cell Line, # SW-626				
	Line					
H0687	Human normal	Human normal	Ovary			pCMVSport 3.0
	ovary(#9610G215)	ovary(#9610G215)				
H0688	Human Ovarian	Human Ovarian				pCMVSport 3.0
	Cancer(#9807G017)	cancer(#9807G017),mRNA from Maura Ru				
H0689	Ovarian Cancer	Ovarian Cancer, #9806G019				pCMVSport 3.0

Code	Description	Tissue	Organ	Cell Line	Disease	Vector	
Н0690	Ovarian Cancer, # 9702G001	Ovarian Cancer, #9702G001				pCMVSport 3.0	
H0691	Normal Ovary, #9710G208	normal ovary, #9710G208				pCMVSport 3.0	
H0693	Normal Prostate #ODQ3958EN	Normal Prostate Tissue # ODQ3958EN				pCMVSport 3.0	
H0694	Prostate gland adenocarcinoma	Prostate gland, adenocarcinoma, mod/diff, gleason	prostate gland			pCMVSport 3.0	
H0695	mononucleocytes from patient	mononucleocytes from patient at Shady Grove Hospit				pCMVSport 3.0	
N0003	Human Fetal Brain	Human Fetal Brain					
9000N	Human Fetal Brain	Human Fetal Brain					
N0007	Human Hippocampus	Human Hippocampus					
6000N	Human Hippocampus, prescreened	Human Hippocampus					
S0001	Brain frontal cortex	Brain frontal cortex	Brain			Lambda ZAP II	
S0002	Monocyte activated	Monocyte-activated	poolq	Cell Line		Uni-ZAP XR	
80003	Human Osteoclastoma	Osteoclastoma	bone		disease	Uni-ZAP XR	<del>,</del>
S0004	Prostate	Prostate BPH	Prostate			Lambda ZAP II	
<b>20009</b>	Neuroblastoma	Human Neural Blastoma			disease	pCDNA	
20007	Early Stage Human Brain	Human Fetal Brain		-		Uni-ZAP XR	
S0010		Amygdala				Uni-ZAP XR	
S0011	STROMAL - OSTEOCLASTOMA	Osteoclastoma	bone		disease	Uni-ZAP XR	
S0013	Prostate	Prostate	prostate			Uni-ZAP XR	
S0014	Kidney Cortex	Kidney cortex	Kidney			Uni-ZAP XR	
S0015	-	Kidney medulla	Kidney			Uni-ZAP XR	
S0016	$\vdash$	Kidney pyramids	Kidney			Uni-ZAP XR	٠,
S0022	Human Osteoclastoma	Osteoclastoma Stromal Cells				Uni-ZAP XR	

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
	Stromal Cells - unamplified				-	
S0024	Human Kidney Medulla - unamplified	Human Kidney Medulla				
S0026	Stromal cell TF274	stromal cell	Bone marrow	Cell Line		Uni-ZAP XR
S0027	Smooth muscle, serum treated	Smooth muscle	Pulmanary artery	Cell Line		Uni-ZAP XR
80028	Smooth muscle, control	Smooth muscle	Pulmanary artery	Cell Line		Uni-ZAP XR
S0029	brain stem	Brain stem	brain			Uni-ZAP XR
S0031	Spinal cord	Spinal cord	spinal cord			Uni-ZAP XR
S0032	Smooth muscle-ILb induced	Smooth muscle	Pulmanary artery	Cell Line		Uni-ZAP XR
S0036	Human Substantia Nigra	Human Substantia Nigra				Uni-ZAP XR
S0037	Smooth muscle, IL1b induced	Smooth muscle	Pulmanary artery	Cell Line		Uni-ZAP XR
80038	Human Whole Brain #2 - Oligo dT > 1.5Kb	Human Whole Brain #2				ZAP Express
80039	Hypothalamus	Hypothalamus	Brain			Uni-ZAP XR
S0040	Adipocytes	Human Adipocytes from Osteoclastoma				Uni-ZAP XR
S0042	Testes	Human Testes				ZAP Express
S0044	Prostate BPH	prostate BPH	Prostate		disease	Uni-ZAP XR
S0045	Endothelial cells- control	Endothelial cell	endothelial cell- lung	Cell Line		Uni-ZAP XR
S0046	Endothelial-induced	Endothelial cell	endothelial cell- lung	Cell Line		Uni-ZAP XR
S0048	Human Hypothalamus, Alzheimer"s	Human Hypothalamus, Alzheimer''s			disease	Uni-ZAP XR
S0049	Human Brain, Striatum	Human Brain, Striatum				Uni-ZAP XR
S0050	Human Frontal Cortex,	Human Frontal Cortex,			disease	Uni-ZAP XR

								0.4	~		~	~	~	~	РΠ	~	~	~	~	~	~
Vector		Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	pBluescript	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Lambda ZAP II	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XR	Uni-ZAP XE
Disease		disease			disease	disease				disease											
Cell Line			Cell Line	Cell Line				Cell Line			Cell Line	Cell Line		Cell Line	Cell Line	Cell Line	Cell Line				Cell Line
Organ			poold	poold	BRAIN	Brain	Brain		Bone marrow	bone	Pulmanary artery	Knee			Bone marrow	gunl	poold		Prostate	prostate	Prostate
Tissue	Schizophrenia	Human Hypothalamus, Schizophrenia	human neutrophils	human neutrophil induced				Anergic T-cell	Bone marrow	Osteoclastoma	Smooth muscle	Osteoblasts	Airway Epithelial	apoptotic cells	stromal cell	eosinophil	macrophage-oxidized LDL treated	Macrophage (GM-CSF treated)	prostate BPH	Prostate	LNCAP Cell Line
Description	Schizophrenia	Human Hypothalmus, Schizoph	neutrophils control	Neutrophils IL-1 and LPS induced	STRIATUM DEPRESSION	Brain Amygdala	Hypothalamus	Anergic T-cell	Bone marrow	Osteoclastoma- normalized A	Smooth muscle-edited	Osteoblasts	Epithelial-TNFa and INF induced	Apoptotic T-cell	PERM TF274	eosinophil-IL5 induced	Macrophage-oxLDL	Macrophage (GM-CSF treated)	prostate-edited	Normal Prostate	LNCAP prostate cell line
Code		S0051	\$0052	<b>†</b>	S0106	20110	\$0112	80114	80116	S0122	S0124	S0126	S0132	S0134	S0136	S0140	S0142	S0144	S0146	S0148	S0150

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
S0152	PC3 Prostate cell line	PC3 prostate cell line				Uni-ZAP XR
80168	Prostate/LNCAP, subtraction I	PC3 prostate cell line				pBluescript
S0176	Prostate, normal, subtraction I	Prostate	prostate			Uni-ZAP XR
S0180	Bone Marrow Stroma, TNF&LPS ind	Bone Marrow Stroma, TNF & LPS induced			disease	Uni-ZAP XR
S0182	Human B Cell 8866	Human B- Cell 8866				Uni-ZAP XR
S0190	Prostate BPH,Lib 2, subtracted	Human Prostate BPH				pSport1
S0192	Synovial Fibroblasts (control)	Synovial Fibroblasts			•	pSport1
S0194	Synovial hypoxia	Synovial Fibroblasts				pSport1
S0196	Synovial IL-1/TNF stimulated	Synovial Fibroblasts				pSport1
S0206	Smooth Muscle- HASTE normalized	Smooth muscle	Pulmanary artery	Cell Line		pBluescript
S0208	Messangial cell, frac 1	Messangial cell				pSport1
S0210	Messangial cell, frac 2	Messangial cell				pSport1
S0212	Bone Marrow Stromal Cell, untreated	Bone Marrow Stromal Cell,untreated				pSport1 .
S0214	Human Osteoclastoma, re-excision	Osteoclastoma	poue		disease	Uni-ZAP XR
S0216	Neutrophils IL-1 and LPS induced	human neutrophil induced	poold	Cell Line		Uni-ZAP XR
S0218	Apoptotic T-cell, re- excision	apoptotic cells		Cell Line		Uni-ZAP XR
S0220	H. hypothalamus, frac A;re-excision	Hypothalamus	Brain			ZAP Express
S0222	H. Frontal cortex,epileptic;re- excision	H. Brain, Frontal Cortex, Epileptic	Brain		disease	Uni-ZAP XR

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
S0242	Synovial Fibroblasts (III/TNF), subt	Synovial Fibroblasts				pSport1
S0250	Human Osteoblasts II	Human Osteoblasts	Femur		disease	pCMVSport 2.0
S0260	Spinal Cord, re- excision	Spinal cord	spinal cord			Uni-ZAP XR
S0276	Synovial hypoxia-RSF subtracted	Synovial fobroblasts (rheumatoid)	Synovial tissue			pSport1
S0278	H Macrophage (GM-CSF treated), re-excision	Macrophage (GM-CSF treated)				Uni-ZAP XR
S0280	Human Adipose Tissue, re-excision	Human Adipose Tissue				Uni-ZAP XR
S0282	Brain Frontal Cortex, re-excision	Brain frontal cortex	Brain			Lambda ZAP II
S0292	Osteoarthritis (OA-4)	Human Osteoarthritic Cartilage	Bone		disease	pSport1
S0294	Larynx tumor	Larynx tumor	Larynx,vocal cord		disease	pSportl
S0298	Bone marrow stroma,treated	Bone marrow stroma,treatedSB	Bone marrow		,	pSport1
80300	Frontal lobe, dementia; re-excision	Frontal Lobe dementia/Alzheimer"s	Brain			Uni-ZAP XR
S0306	Larynx normal #10 261-273	Larynx normal		-		pSport1
S0312	Human osteoarthritic;fraction II	Human osteoarthritic cartilage			disease	pSport1
S0314	Human osteoarthritis;fraction I	Human osteoarthritic cartilage			disease	pSport1
S0316	Human Normal Cartilage, Fraction I	Human Normal Cartilage				pSport1

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
\$0318	Human Normal	Human Normal Cartilage				pSport1
	Cartilage Fraction II					
S0328	Palate carcinoma	Palate carcinoma	Uvula		disease	pSport1
S0330	Palate normal	Palate normal	Uvula			pSport1
S0332	Pharynx carcinoma	Pharynx carcinoma	Hypopharynx			pSport1
S0334	Human Normal	Human Normal Cartilage			-	pSport1
	Cartilage Fraction III		-			
S0336	Human Normal	Human Normal Cartilage				pSport1
	Cartilage Fraction IV					
80338	Human Osteoarthritic	Human osteoarthritic			disease	pSport1
00240	Human Ostanarthritic	Human osteoarthritic			disease	pSport1
04505	Cartilage Fraction IV	cartilage				
S0342	Adipocytes;re-excision	Human Adipocytes from Osteoclastoma				Uni-ZAP XR
S0344	Macrophage-oxLDL; re-excision	macrophage-oxidized LDL treated	plood	Cell Line		Uni-ZAP XR
S0346	Human Amygdala;re-	Amygdala				Uni-ZAP XR
	excision					
S0348	Cheek Carcinoma	Cheek Carcinoma			disease	pSport1
80350	Pharynx Carcinoma	Pharynx carcinoma	Hypopharynx		disease	pSport1
S0352	Larynx Carcinoma	Larynx carcinoma			disease	pSport1
S0354	Colon Normal II	Colon Normal	Colon			pSport1
S0356	Colon Carcinoma	Colon Carcinoma	Colon	-	disease	pSport1
S0358	Colon Normal III	Colon Normal	Colon			pSport1
S0360	Colon Tumor II	Colon Tumor	Colon		disease	pSport1
S0362	Human Gastrocnemius	Gastrocnemius muscle				pSport1
S0364	Human Quadriceps	Quadriceps muscle				pSport1
S0366	Human Soleus	Soleus Muscle				pSport1
80368	Human Pancreatic	Islets of Langerhans				pSport1
S0372	Larynx carcinoma III	Larynx carcinoma			disease	pSport1

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
S0374	Normal colon	Normal colon				pSport1
S0376	Colon Tumor	Colon Tumor			disease	pSport1
S0378	Pancreas normal PCA4 No	Pancreas Normal PCA4 No				pSport1
S0380	Pancreas Tumor PCA4 Tu	Pancreas Tumor PCA4 Tu			disease	pSport1
S0382	Larynx carcinoma IV	Larynx carcinoma			disease	pSport1
S0384	Tongue carcinoma	Tongue carcinoma			disease	pSport1
S0386	Human Whole Brain, re-excision	Whole brain	Brain			ZAP Express
S0388	Human .	Human Hypothalamus,			disease	Uni-ZAP XR
	Hypothalamus, schizop hrenia, re-excision	Schizophrenia				
80390	Smooth muscle,	Smooth muscle	Pulmanary	Cell Line		Uni-ZAP XR
	control; re-excision		artery			
S0392	Salivary Gland	Salivary gland; normal				pSport1
S0394	Stomach; normal	Stomach; normal				pSport1
S0398	Testis; normal	Testis; normal				pSport1
S0402	Adrenal Gland, normal	Adrenal gland; normal				pSport1
S0404	Rectum normal	Rectum, normal				pSport1
S0406	Rectum tumour	Rectum tumour				pSport1
S0408	Colon, normal	Colon, normal				pSport1
S0410	Colon, tumour	Colon, tumour				pSport1
S0412	Temporal cortex-	Temporal cortex, alzheimer			disease	Other
	Alzheizmer; subtracted					
S0414	Hippocampus, Alzheimer Subtracted	Hippocampus, Alzheimer Subtracted				Other
S0418	CHIME Cell	CHME Cell Line; treated				pCMVSport 3.0
	Line;treated 5 hrs					
S0420	CHME Cell Line, untreated	CHME Cell line, untreatetd				pSport1
S0422	Mo7e Cell Line GM-	Mo7e Cell Line GM-CSF				pCMVSport 3.0

Description	Tissue	Organ	Cell Line	Disease	Vector
trea	treated (1ng/ml)				
TF-1 Cel	TF-1 Cell Line GM-CSF Treated				pSport1
Monoc	Monocyte-activated	poold	Cell Line		Uni-ZAP XR
human	human neutrophils	blood	Cell Line		Uni-ZAP XR
Aryepig	Aryepiglottis Normal				pSport1
Sinus pinit	Sinus piniformis Tumour				pSport1
Stoma	Stomach Normal			disease	pSport1
Stomac	Stomach Tumour			disease	pSport1
Liver Nor	Liver Normal Met5No				pSport1
Liver	er Tumour				pSport1
Color	Colon Normal				pSport1
Colon	Colon Tumour			disease	pSport1
Tongn	Tongue Tumour				pSport1
Laryn	Larynx Normal				pSport1
Laryn	Larynx Tumour				pSport1
T	Thymus				pSport1
Pl	Placenta	Placenta			pSport1
Tongı	Tongue Normal				pSport1
Thyrc	Thyroid normal				pSport1
Thyro	id Tumour				pSport1
Thyroic	Thyroid Thyroiditis				pSport1
Lary	Larynx Normal				pSport1
Lary	Larynx Tumor			disease	pSport1
Ea.hy	Ea.hy.926 cell line				pSport1
	PYFD			disease	pSport

S0474 S0665					ACMOS A	1000
	Human blood platelets	Platelets	Blood platelets			Other
	Human Amygdala; re- excission	Amygdala				Uni-ZAP XR
S3012	Smooth Muscle Serum Treated, Norm	Smooth muscle	Pulmanary artery	Cell Line		pBluescript
S3014	Smooth muscle, serum induced,re-exc	Smooth muscle	Pulmanary artery	Cell Line		pBluescript
S6014	H. hypothalamus, frac A	Hypothalamus	Brain			ZAP Express
S6016	H. Frontal Cortex, Epileptic	H. Brain, Frontal Cortex, Epileptic	Brain		disease	Uni-ZAP XR
S6022	H. Adipose Tissue	Human Adipose Tissue				Uni-ZAP XR
S6024	Alzheimers, spongy change	Alzheimer"s/Spongy change	Brain		disease	Uni-ZAP XR
S6026	Frontal Lobe, Dementia	Frontal Lobe dementia/Alzheimer"s	Brain			Uni-ZAP XR
S6028	Human Manic Depression Tissue	Human Manic depression tissue	Brain		disease	Uni-ZAP XR
T0002	Activated T-cells	Activated T-Cell, PBL fraction	Blood	Cell Line		pBluescript SK-
T0003	Human Fetal Lung	Human Fetal Lung				pBluescript SK-
T0004	Human White Fat	Human White Fat				pBluescript SK-
T0006	Human Pineal Gland	Human Pinneal Gland				pBluescript SK-
T0008	Colorectal Tumor	Colorectal Tumor			disease	pBluescript SK-
T0010	Human Infant Brain	Human Infant Brain				Other
T0023	Human Pancreatic Carcinoma	Human Pancreatic Carcinoma			disease	pBluescript SK-
T0039	HSA 172 Cells	Human HSA172 cell line				pBluescript SK-
T0040	HSC172 cells	SA172 Cells				pBluescript SK-
T0041	Jurkat T-cell G1 phase	Jurkat T-cell				pBluescript SK-
T0042	Jurkat T-Cell, S phase	Jurkat T-Cell Line				pBluescript SK-
T0048	Human Aortic	Human Aortic Endothilium				pBluescript SK-

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
	Endothelium					
T0049	Aorta endothelial cells + TNF-a	Aorta endothelial cells				pBluescript SK-
T0060	Human White Adipose	Human White Fat				pBluescript SK-
T0067	Human Thyroid	Human Thyroid				pBluescript SK-
T0068	Normal Ovary,	Normal Ovary,				pBluescript SK-
700060	Human Henis normal	Human [ Itemis normal				pBluescript SK-
T0071	Human Bone Marrow	Human Bone Marrow				pBluescript SK-
T0082	Human Adult Retina					pBluescript SK-
T0087	Alzheimer"s, exon trap,712P				disease	pAMP
T0103	Human colon					pBluescript SK-
	carcinoma (HCC) cell line					
T0104	HCC cell line					pBluescript SK-
	metastisis to liver					
T0109	Human (HCC) cell line					pBluescript SK-
	liver (mouse) metastasis, remake					
T0110	Human colon					pBluescript SK-
	carcinoma (HCC) cell line, remake					
T0112	Human (Caco-2) cell			•		pBluescript SK-
	line, adenocarcinoma,					
T0114	Human (Caco-2) cell					pBluescript SK-
	line, adenocarcinoma,					
T0115	Human Colon					pBluescript SK-
	Carcinoma (HCC) cell					
	71117					

Vector																					
Disease																					
Cell Line																			,		
Organ											•										
Tissue																		amygdala	aorta	placenta	placenta
Description	Atrium cDNA library	Clontech human aorta	polyA+ mRNA (#6572)	Human (M.Lovett)	Human adult (K.Okubo)	Human adult lung 3" directed Mbol cDNA	Human brain ARSanders	Human colon mucosa	Human epidermal keratinocyte	Human keratinocyte	differential display (B.Lin)	Human pancreatic	Human promyelocyte	Human thymus NSTH	Liver HepG2 cell line.	Selected chromosome	21 cDNA library	DKFZphamy1	Human aorta polyA+ (TFujiwara)	Human placenta cDNA (TFujiwara)	Human placenta
Code		T0002		L0018	L0021	L0022	L0024	L0040	L0041	L0045		L0053	L0055	<del></del>	L0065	┼	-+	_	T0102	L0142	L0143

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Vector							BA, M13-derived	BA, M13-derived		Bluescript	Bluescrint	J	Bluescript SK	Bluescript SK-	Bluescript SK-	Bluescript SK-	Bluescript SK-	Jugarine CV	Diuescript on-	Bluescript SK-	Bluescript SK-	Bluescript SK-	Bluescript SK-	Bluescript SK-
Λ							<u> </u>	B				3	Э.	ш-	1	I		   		41	1		1	
Disease																								
Cell Line		-		HeLa	Patu 8988t																			
Cell					H			-													pu			
Organ		brain	heart										ovary								adrenal gland	brain	breast	colon
Tissue					pancreatic cancer										germ cell tumor	germ cell tumor	schizophrenic brain S-11	frontal lobe	Schwannoma tumor	synovial sarcoma	adrenal adenoma	pooled frontal lobe	breast tumor	colon tumor
Description	polyA+ (TFujiwara)	Human fetal brain (TFujiwara)	Human heart cDNA	Human HeLa cells	Human pancreatic	8988t	Infant brain, Bento	Normalized infant	brain, Bento Soares	P, Human foetal Brain	whole tissue	S, Human foetal	Stratagene ovary (#937217)	Stratagene ovarian	NCI CGAP GC2	NCI CGAP GC5	Stratagene schizo brain	SIII	NCI_CGAP_Sch1	NCI_CGAP_SS1	NCI CGAP AA1	Johnston frontal cortex	NCI CGAP_Br3	
Code	╌	L0157	L0163	L0183	L0194		L0351	L0352		L0355	,	L0356	L0361	L0362	1 0363	10364	L0366	_	L0367	L0368	L0369	L0370	170371	

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
╁╴	NCI_CGAP_Col1	tumor	colon			Bluescript SK-
L0374	NCI_CGAP_Co2	tumor	colon			Bluescript SK-
-	NCI_CGAP_Kid6	kidney tumor	kidney			Bluescript SK-
L0376	NCI_CGAP_Lar1	larynx	larynx			Bluescript SK-
L0378	NCI_CGAP_Lu1	lung tumor	lung			Bluescript SK-
L0379	NCI_CGAP_Lym3	lymphoma	lymph node			Bluescript SK-
├	NCI CGAP HN4	squamous cell carcinoma	pharynx			Bluescript SK-
┢	NCI_CGAP_Pr25	epithelium (cell line)	prostate			Bluescript SK-
L0383	NCI CGAP Pr24	invasive tumor (cell line)	prostate			Bluescript SK-
L0384	NCI_CGAP_Pr23	prostate tumor	prostate			Bluescript SK-
├	NCI_CGAP_Gas1	gastric tumor	stomach			Bluescript SK-
F0386	NCI_CGAP_HIN3	squamous cell carcinoma	tongue			Bluescript SK-
_		from base of tongue				
L0387	NCI_CGAP_GCB0	germinal center B-cells	tonsil			Bluescript SK-
	NCI_CGAP_HN6	normal gingiva (cell line from immortalized kerati				Bluescript SK-
L0389	NCI_CGAP_HN5	normal gingiva (cell line from primary keratinocyt				Bluescript SK-
L0394	H, Human adult Brain Cortex tissue					gt11
L0404	b4HB3MA Cot109+103+85-Bio					Lafmid A
L0411	1-NB					Lafmid BA
L0415	b4HB3MA Cot8-HAP- Ft					Lafmid BA
L0418	b4HB3MA- Cot109+10-Bio					Lafmid BA
L0428	Cot1374Ft-4HB3MA					Lafmid BA
L0434	Infant brain library of Dr. M. Soares					lafmid BA
L0435	Infant brain, LLNL array of Dr. M. Soares			·		lafmid BA

Code	Description	Tissue	Organ	Cell Line	Disease	Vector	
	INIB						
L0438	normalized infant brain cDNA	total brain	brain			lafmid BA	
L0439	Soares infant brain INIB		whole brain			Lafmid BA	
L0443	b4HB3MK					Lafmid BK	
L0454	Clontech adult human fat cell library					lambda gt10	
L0455	Human retina cDNA randomly primed	retina	eye			lambda gt10	
L0456	Human retina cDNA Tsp5091-cleaved	retina	eye			lambda gt10	
L0459	Adult heart, Clontech					Lambda gt11	
L0460	Adult heart, Lambda gt 11					Lambda gt11	
L0462	WATMI					lambda gt11	
L0463	fetal brain cDNA	brain	brain			lambda gt11	
L0471	Human fetal heart, Lambda ZAP Express					Lambda ZAP Express	
L0475	KG1-a Lambda Zap Express cDNA library			KG1-a		Lambda Zap Express (Stratagene)	
L0476	Fetal brain, Stratagene		•			Lambda ZAP II	
L0480	Stratagene cat#937212 (1992)					Lambda ZAP, pBluescript SK(-)	
L0481	CD34+DIRECTIONA L				-	Lambda ZAPII	
L0483	Human pancreatic islet					Lambda ZAPII	
L0485	STRATAGENE Human skeletal muscle	skeletal muscle	leg muscle			Lambda ZAPII	

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
	cDNA library, cat. #936215.					
L0492	Human Genomic					pAMP ,
1	NCI_CGAP_0v26	papillary serous carcinoma	ovary			pAMP1
	NCI_CGAP_HSC4	CD34+, CD38- from normal	ропе татом			pAMP1
-		bone marrow donor				
L0499	NCI_CGAP_HSC2	stem cell 34+/38+	bone marrow			pAMP1
L0500	NCI_CGAP_Bm20	oligodendroglioma	brain			pAMP1
L0506	NCI_CGAP_Br16	lobullar carcinoma in situ	breast			pAMP1
L0509	NCI_CGAP_Lu26	invasive adenocarcinoma	lung			pAMP1
┿	NCI_CGAP_0v33	borderline ovarian carcinoma	ovary			pAMP1
L0511	NCI_CGAP_0v34	borderline ovarian carcinoma	ovary			pAMP1
L0512	NCI_CGAP_0v36	borderline ovarian carcinoma	ovary			pAMP1
1.0514	NCI_CGAP_0v31	papillary serous carcinoma	ovary			pAMP1
L0515	NCI_CGAP_0v32	papillary serous carcinoma	ovary			pAMP1
L0517	NCI_CGAP_Pr1		•			pAMP10
L0518	NCI_CGAP_Pr2					pAMP10
<del></del>	NCI_CGAP_Pr3					pAMP10
L0520	NCI_CGAP_Alv1	alveolar rhabdomyosarcoma				pAMP10
L0521	NCI_CGAP_Ew1	Ewing"s sarcoma				pAMP10
	NCI_CGAP_Kid1	kidney				pAMP10
L0523	NCI_CGAP_Lip2	liposarcoma				pAMP10
. L0526	NCI_CGAP_Pr12	metastatic prostate bone lesion		-		pAMP10
L0527	NCI_CGAP_0v2	ovary				pAMP10
L0528	NCI_CGAP_Pr5	prostate				pAMP10
L0529	NCI_CGAP_Pr6	prostate				pAMP10
L0530	NCI_CGAP_Pr8	prostate				pAMP10
L0532	NCI_CGAP_Thy1	thyroid				pAMP10
L0534	Chromosome 7 Fetal	brain	brain			pAIMP10
	Diam כעונט Diamy					

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
L0539	Chromosome 7		placenta			pAMP10
	Placental cDNA Library	•				
L0540	NCI_CGAP_Pr10	invasive prostate tumor	prostate			pAMP10
	NCI_CGAP_Pr11	normal prostatic epithelial cells	prostate			pAMP10
L0543	NCI_CGAP_Pr9	normal prostatic epithelial cells	prostate			pAMP10
L0544	NCI_CGAP_Pr4	prostatic intraepithelial neoplasia - high grade	prostate			pAMP10
L0545	NCI_CGAP_Pr4.1	prostatic intraepithelial neoplasia - high grade	prostate			pAMP10
L0547	NCI_CGAP_Pr16	tumor	prostate			pAMP10
_	NCI_CGAP_Co22	colonic adenocarcinoma	colon			pAMP10
L0558	NCI_CGAP_Ov40	endometrioid ovarian metastasis	ovary			pAMP10
L0559	NCI_CGAP_Ov39	papillary serous ovarian metastasis	ovary			pAMP10
T0560	L0560 NCI_CGAP_HN12	moderate to poorly differentiated invasive carcino	tongue			pAMP10
L0561	NCI_CGAP_HN11	normal squamous epithelium	tongue			pAMP10
L0562	Chromosome 7 HeLa cDNA Library			HeLa cell line; ATCC	,	pAMP10
L0564	Jia bone marrow stroma	bone marrow stroma				pBluescript
L0565	Normal Human Trabecular Bone Cells	Bone	Hip			pBluescript
L0581	Stratagene liver (#937224)		liver			pBluescript SK
T0586	HTCDLI					pBluescript SK(-)
L0587	Stratagene colon HT29					pBluescript SK-

	Description	Tissue	<u>Organ</u>	Cell Line	Disease	Vector
(#63,	(#937221)					
Stra cell	Stratagene endothelial cell 937223					pBluescript SK-
Str 93	Stratagene fetal retina 937202					pBluescript SK-
あき	Stratagene fibroblast (#937212)					pBluescript SK-
S, S	Stratagene HeLa cell s3 937216					pBluescript SK-
ت مرا	Stratagene hNT neuron (#937233)					pBluescript SK-
103	Stratagene					pBluescript SK-
	neuroepithelium (#937231)					
روا	Stratagene					pBluescript SK-
	neuroepithelium NT2RAMI 937234		•			
٠,	Stratagene NT2	neuroepithelial cells	brain			pBluescript SK-
H 0	neuronal precursor 937230				:	
	Stratagene colon (#937204)		colon			pBluescript SK-
מ נטן	Stratagene corneal stroma (#937222)		comea			pBluescript SK-
~	Morton Fetal Cochlea	cochlea	ear			pBluescript SK-
0,0	Stratagene lung (#937210)		lung			pBluescript SK-
I	Weizmann Olfactory Epithelium	olfactory epithelium	nose			pBluescript SK-
5	Stratagene pancreas (#937208)		pancreas			pBluescript SK-
	Pancreatic Islet	pancreatic islet	pancreas			pBluescript SK-

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
L0603	Stratagene placenta (#937225)		placenta			pBluescript SK-
L0604	Stratagene muscle 937209	muscle	skeletal muscle			pBluescript SK-
T0605	Stratagene fetal spleen (#937205)	fetal spleen	uəəlds			pBluescript SK-
T0606	NCI_CGAP_Lym5	follicular lymphoma	lymph node			pBluescript SK-
L0607	NCI_CGAP_Lym6	mantle cell lymphoma	lymph node			pBluescript SK-
F0608	Stratagene lung carcinoma 937218	lung carcinoma	lung	NCI-H69		pBluescript SK-
F0000	Schiller astrocytoma	astrocytoma	brain			pBluescript SK- (Stratagene)
L0611	Schiller meningioma	meningioma	brain			pBluescript SK- (Stratagene)
L0612	Schiller olivodendroglioma	oligodendroglioma	brain			pBluescript SK- (Stratagene)
L0615	22 week old human fetal liver cDNA					pBluescriptII SK(-)
	library					
L0616	Chromosome 21 exon					pBluescriptIIKS+
L0617	Chromosome 22 exon					pBluescriptIffS+
L0622	HMI					pcDNAII (Invitrogen)
L0623	HM3	pectoral muscle (after mastectomy)				pcDNAII (Invitrogen)
L0625	NCI_CGAP_AR1	bulk alveolar tumor				pCMV-SPORT2
T0626	NCI_CGAP_GC1	bulk germ cell seminoma				pCMV-SPORT2
L0627	NCI_CGAP_Co1	bułk tumor	colon			pCMV-SPORT2
L0628	NCI_CGAP_0v1	ovary bulk tumor	ovary			pCMV-SPORT2
L0629	NCI_CGAP_Mel3	metastatic melanoma to	bowel (skin			pCMV-SPORT4
1.0620	NOT COAD CNC1	curbetantia nigra	hrain			DCMV-SPORT4
10001	NOT COAD B-7	Substantia ingla	hreact			nCMV-SPORT4
10021	NCI CGAP_BT/		UICASI			POINT STORY

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
L0632	NCI CGAP_Li5	hepatic adenoma	liver			pCMV-SPORT4
L0633	NĊI_CGAP_Lu6	small cell carcinoma	Jung			pCMV-SPORT4
L0634	NCI_CGAP_Ov8	serous adenocarcinoma	ovary			pCMV-SPORT4
L0635	NCI_CGAP_PNS1	dorsal root ganglion	peripheral			pCMV-SPORT4
			nervous system			
T0636	NCI_CGAP_Pit1	four pooled pituitary	brain			pCMV-SPORT6
L0637	NCI CGAP Bm53	three pooled meningiomas	brain			pCMV-SPORT6
L0638	NCI_CGAP_Brn35	tumor, 5 pooled (see description)	brain			pCMV-SPORT6
L0639	NCI_CGAP_Brn52	tumor, 5 pooled (see description)	brain			pCMV-SPORT6
L0640	NCI_CGAP_Br18	four pooled high-grade tumors, including two prima	breast			pCMV-SPORT6
L0641	NCI_CGAP_Co17	juvenile granulosa tumor	colon			pCMV-SPORT6
L0642	NCI_CGAP_Co18	moderately differentiated adenocarcinoma	colon			pCMV-SPORT6
L0643	NCI_CGAP_Co19	moderately differentiated adenocarcinoma	colon			pCMV-SPORT6
L0644	NCI_CGAP_Co20	moderately differentiated adenocarcinoma	colon			pCMV-SPORT6
L0645	NCI_CGAP_Co21	moderately differentiated adenocarcinoma	colon			pCMV-SPORT6
L0646	NCI_CGAP_Co14	moderately-differentiated adenocarcinoma	colon	-		pCMV-SPORT6
L0647	NCI_CGAP_Sar4	five pooled sarcomas, including myxoid liposarcoma	connective tissue			pCMV-SPORT6
L0648	NCI_CGAP_Eso2	squamous cell carcinoma	esophagus			pCMV-SPORT6
L0649	NCI_CGAP_GU1	2 pooled high-grade transitional cell tumors	genitourinary tract			pCMV-SPORT6
L0650	NCI_CGAP_Kid13	2 pooled Wilms" tumors, one	kidney			pCMV-SPORT6

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
		primary and one metast				
L0651	NCI_CGAP_Kid8	renal cell tumor	kidney			pCMV-SPORT6
L0652	NCI_CGAP_Lu27	four pooled poorly- differentiated adenocarcinomas	lung			pCMV-SPORT6
L0653	NCI_CGAP_Lu28	two pooled squamous cell carcinomas	gunl			pCMV-SPORT6
L0654	NCI_CGAP_Lu31		lung, cell line			pCMV-SPORT6
	NCI_CGAP_Lym12	lymphoma, follicular mixed small and large cell	lymph node			pCMV-SPORT6
T0656	NCI_CGAP_0v38	normal epithelium	ovary			pCMV-SPORT6
L0657	NCI_CGAP_0v23	tumor, 5 pooled (see description)	ovary			pCMV-SPORT6
L0658	NCI_CGAP_0v35	tumor, 5 pooled (see description)	очагу			pCMV-SPORT6
L0659	NCI_CGAP_Pan1	adenocarcinoma	pancreas			pCMV-SPORT6
L0661	NCI_CGAP_Mel15	malignant melanoma, metastatic to lymph node	skin			pCMV-SPORT6
T0662	NCI_CGAP_Gas4	poorly differentiated adenocarcinoma with signet r	stomach			pCMV-SPORT6
L0663	NCI_CGAP_Ut2	moderately-differentiated endometrial adenocarcino	uterus			pCMV-SPORT6
L0664	NCI_CGAP_Ut3	poorly-differentiated endometrial adenocarcinoma,	uterus			pCMV-SPORT6
T0665	NCI_CGAP_Ut4	serous papillary carcinoma, high grade, 2 pooled t	uterus			pCMV-SPORT6
T0666	NCI_CGAP_Ut1	well-differentiated endometrial adenocarcinoma,	uterus			pCMV-SPORT6
T0992	NCI_CGAP_CML1	myeloid cells, 18 pooled CML cases, BCR/ABL rearra	whole blood			pCMV-SPORT6
L0683	Stanley Frontal NS	frontal lobe (see description)	brain			pCR2.1-TOPO (Invitrogen)

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
	pool 2					
T0686	Stanley Frontal SN pool 2	frontal lobe (see description)	brain			pCR2.1-TOPO (Invitrogen)
F0698	Testis 2					PGEM 5zf(+)
L0709	NIH_MGC_21	choriocarcinoma	placenta			pOTB7
L0710	NIH_MGC_7	small cell carcinoma	gunl	MGC3		pOTB7
L0717	Gessler Wilms tumor					pSPORT1
L0718	Testis 5					pSPORT1
L0731	Soares_pregnant_uteru s_NbHPU		uterus			pT7T3-Pac
L0738	Human colorectal					pT7T3D
L0740	Soares melanocyte 2NbHM	melanocyte				pT7T3D (Pharmacia) with a modified polylinker
L0741	Soares adult brain N2b4HB55Y		brain			pT7T3D (Pharmacia) with a modified polylinker
L0742	Soares adult brain N2b5HB55Y		brain			pT7T3D (Pharmacia) with a modified polylinker
L0743	Soares breast 2NbHBst		breast			pT7T3D (Pharmacia) with a modified polylinker
L0744	Soares breast 3NbHBst		breast			pT7T3D (Pharmacia) with a modified polylinker
L0745	Soares retina N2b4HR	retina	eye			pT/T3D (Pharmacia) with a modified polylinker
L0746	Soares retina N2b5HR	retina	eye			pT7T3D (Pharmacia) with a modified polylinker
L0747	Soares_fetal_heart_Nb HH19W		heart			pT7T3D (Pharmacia) with a modified polylinker
L0748	Soares fetal liver spleen 1NFLS		Liver and Spleen			pT7T3D (Pharmacia) with a modified polylinker
L0749	Soares_fetal_liver_sple en_INFLS_S1		Liver and Spleen			pT7T3D (Pharmacia) with a modified polylinker

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
L0750	Soares_fetal_lung_Nb		lung			pT7T3D (Pharmacia) with
	HL19W					a modified polylinker
L0751	Soares ovary tumor	ovarian tumor	ovary			pT7T3D (Pharmacia) with
	NeHOI					a mounted polymines
L0752	Soares_parathyroid_tu	parathyroid tumor	parathyroid			pT7T3D (Pharmacia) with
	mor_NbHPA		gland			a modified polylinker
L0753	Soares_pineal_gland_		pineal gland			pT7T3D (Pharmacia) with
	N3HPG					a modified polylinker
L0754	Soares placenta		placenta			pT7T3D (Pharmacia) with
	Nb2HP					a modified polylinker
L0755	Soares_placenta_8to9		placenta			pT7T3D (Pharmacia) with
	weeks_2NbHP8to9W					a modified polylinker
10756	Soares_multiple_sclero	multiple sclerosis lesions				pT7T3D (Pharmacia) with
	sis 2NbHMSP	•				a modified polylinker
	•					V_TYPE
L0757	Soares_senescent_fibro	senescent fibroblast				pT7T3D (Pharmacia) with
	blasts_NbHSF					a modified polylinker
						V_TYPE
1.0758	Soares_testis_NHT					pT7T3D-Pac (Pharmacia)
						with a modified polylinker
L0759	Soares_total_fetus_Nb					pT7T3D-Pac (Pharmacia)
	2HF8_9w					with a modified polylinker
T0760	Barstead aorta	aorta				pT7T3D-Pac (Pharmacia)
	HPLRB3					with a modified polylinker
L0761	NCI_CGAP_CLL1	B-cell, chronic lymphotic				pT7T3D-Pac (Pharmacia)
		leukemia				with a modified polylinker
L0762	NCI_CGAP_Br1.1	breast				pT7T3D-Pac (Pharmacia)
						with a modified polylinker
L0763	NCI_CGAP_Br2	breast				pT7T3D-Pac (Pharmacia)
						with a modified polylinker
L0764	NCI_CGAP_Co3	colon				pT7T3D-Pac (Pharmacia)
						with a modified polyfinker

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
╫	NCI_CGAP_Co4	colon				pT7T3D-Pac (Pharmacia)
-						with a mounted polymine
T0266	NCI_CGAP_GCB1	germinal center B cell				p1/13D-Fac (Pharmacia) with a modified polylinker
1 0767	NCI CGAP GC3	nooled germ cell tumors				pT7T3D-Pac (Pharmacia)
	700-	Toronto de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la companya de la company				with a modified polylinker
T0768	NCI_CGAP_GC4	pooled germ cell tumors				pT7T3D-Pac (Pharmacia) with a modified polylinker
F0769	NCI_CGAP_Bm25	anaplastic oligodendroglioma	brain			pT7T3D-Pac (Pharmacia)
						with a modified polylinker
L0770	NCI_CGAP_Bm23	glioblastoma (pooled)	brain			pT7T3D-Pac (Pharmacia)
						with a modified polyfinker
L0771	NCI_CGAP_Co8	adenocarcinoma	colon			pT7T3D-Pac (Pharmacia)
	ı					with a modified polylinker
L0772	NCI CGAP Co10	colon tumor RER+	colon			pT7T3D-Pac (Pharmacia)
	1					with a modified polylinker
L0773	NCI CGAP Co9	colon tumor RER+	colon			pT7T3D-Pac (Pharmacia)
	i					with a modified polylinker
L0774	NCI CGAP Kid3		kidney			pT7T3D-Pac (Pharmacia)
	1					with a modified polylinker
L0775	NCI_CGAP_Kid5	2 pooled tumors (clear cell	kidney			pT7T3D-Pac (Pharmacia)
		type)				with a modified polylinker
10776	NCI_CGAP_Lu5	carcinoid	gunl			pT7T3D-Pac (Pharmacia)
				•		with a modified polylinker
1.0777	Soares_NhHMPu_S1	Pooled human melanocyte,	mixed (see			pT7T3D-Pac (Pharmacia)
	1	fetal heart, and pregnant	below)			with a modified polylinker
L0778	Barstead pancreas		pancreas			pT7T3D-Pac (Pharmacia)
	HPLRBI		_			with a modified polylinker
L0779	Soares_NFL_T_GBC_		pooled			pT7T3D-Pac (Pharmacia)
	S1					with a modified polylinker
L0780	Soares_NSF_F8_9W_		pooled	-		pT7T3D-Pac (Pharmacia)
	OT_PA_P_S1					with a modified polylinker

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
╁╾	NCI_CGAP_Pr21	normal prostate	prostate			pT7T3D-Pac (Pharmacia)
L0783	NCI_CGAP_Pr22	normal prostate	prostate			pT7T3D-Pac (Pharmacia)
						with a modified polylinker
L0784	NCI_CGAP_Lei2	leiomyosarcoma	soft tissue			pT7T3D-Pac (Pharmacia) with a modified polylinker
1.0785	Barstead spleen		spleen			pT7T3D-Pac (Pharmacia)
3	HPLRB2		-			with a modified polylinker
T0786	Soares_NbHFB		whole brain			pT7T3D-Pac (Pharmacia)
						with a modified polylinker
L0787	NCI_CGAP_Sub1					pT7T3D-Pac (Pharmacia)
						with a mounted polyminer
L0788	NCI_CGAP_Sub2					pi /i sD-rac (rijanijacia) with a modified polylinker
1 0780	NCI CGAP Sub3					pT/T3D-Pac (Pharmacia)
						with a modified polylinker
L0790	NCI CGAP Sub4					pT7T3D-Pac (Pharmacia)
	1					with a modified polylinker
10791	NCI_CGAP_Sub5					pT7T3D-Pac (Pharmacia)
	l					with a modified polylinker
L0792	NCI_CGAP_Sub6					pT7T3D-Pac (Pharmacia)
						with a modified polylinker
L0793	NCI_CGAP_Sub7					pT7T3D-Pac (Pharmacia)
						with a modified polylinker
L0794	NCI_CGAP_GC6	pooled germ cell tumors				pT7T3D-Pac (Pharmacia)
-						with a modified polylinker
70796	NCI_CGAP_Bm50	medulloblastoma	brain			pT7T3D-Pac (Pharmacia)
						with a modified polylinker
T0800	NCI_CGAP_Co16	colon tumor, RER+	colon			pT7T3D-Pac (Pharmacia)
						TTTTT D. (D.
L0803	NCI_CGAP_Kid11		kidney			p1/13D-Fac (Fharmacia) with a modified polylinker

	Description	Tissue	Organ	Cell Line	Disease	Vector
L0804	NCI_CGAP_Kid12	2 pooled tumors (clear cell	kidney			pT7T3D-Pac (Pharmacia)
		type)				with a modified polylinker
L0805	NCI_CGAP_Lu24	carcinoid	gunl			pT7T3D-Pac (Pharmacia)
						with a modified polyfinker
F0806	NCI_CGAP_Lu19	squamous cell carcinoma,	lung			pT7T3D-Pac (Pharmacia)
		poorly differentiated (4				with a modified polylinker
L0807	NCI_CGAP_Ov18	fibrotheoma	ovary			pT7T3D-Pac (Pharmacia)
	1					with a modified polylinker
L0809	NCI_CGAP_Pr28		prostate			pT7T3D-Pac (Pharmacia)
						with a modified polylinker
L0811	BATM2					PTZ18
L0879	┰		breast			puc18
L0946	BT0333		breast			puc18
L1441	CT0249		colon			puc18
L1499	CT0322		colon			puc18
L1788	HT0229		head_neck			puc18
L1877	HT0340		head_neck			puc18
L1878	⊢		head_neck			puc18
L1942	HT0452		head_neck			puc18
L2138	ST0186		stomach			puc18
L2174	$\vdash$		stomach			puc18
L2251	Human fetal lung	Fetal lung				
L2252	Human placenta	placenta				
L2255	-	corresponding non cancerous				pBluescript sk(-)
1,00	+	onsen lavit	12/00			CMV SPOPTK
7577	$\dashv$	adenocarcinoma	COLOII			PCIVI V-31 ON 10
L2258	NIH_MGC_67	retinoblastoma	eye			pCMV-SPORT6
L2259	NIH_MGC_68	large cell carcinoma	lung			pCMV-SPORT6
L2260	NIH_MGC_69	large cell carcinoma, undifferentiated	lung			pCMV-SPORT6
L2261	NIH_MGC_70	epithelioid carcinoma	pancreas			pCMV-SPORT6

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
L2262	NIH_MGC_72	melanotic melanoma	skin			pCMV-SPORT6
L2263	NIH_MGC_66	adenocarcinoma	ovary			pCMV-SPORT6
L2264	NIH_MGC_71	leiomyosarcoma	uterus			pCMV-SPORT6
1	NIH_MGC_39	adenocarcinoma	pancreas			pOTB7
L2270	Lupski_dorsal_root_ga	dorsal root ganglia				pCMV-SPORT6 (Life
	nglion					l'echnologies)
L2333	CT0417		colon			puc18
L2338	CT0432		colon			puc18
L2346	CT0483		colon			puc18
L2367	UT0039		uterus_tumor			puc18
L2400	NN0116		nervous_normal			puc18
L2413	NN0141		nervous_normal			puc18
L2439	NN 1022		nervous_normal			puc18
L2477	HT0408		head_neck			puc18
L2490	HT0545		head_neck			puc18
L2495	HT0594		head_neck			puc18
L2497	HT0618		head_neck			puc18
L2504	HT0636		head_neck			puc18
L2522	HT0704		head_neck			puc18
L2540	HT0728		head_neck			puc18
L2550	HT0743		head_neck			puc18
L2562	HT0760		head_neck			puc18
L2570	HT0771		head_neck			puc18
L2598	HT0809		head_neck	-		puc18
L2634	HT0872		head_neck			puc18
L2637	HT0877		head_neck			puc18
L2647	HT0894		head_neck			puc18
L2651	NIH_MGC_20	melanotic melanoma	skin			pOTB7
L2653	NIH_MGC_58	hypernephroma	kidney			pDNR-LIB (Clontech)
L2654	NIH_MGC_9	adenocarcinoma cell line	ovary			pOTB7
L2655	NIH_MGC_55	from acute myelogenous	bone marrow			pDNR-LIB (Clontech)

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
		leukemia				
L2657	NIH_MGC_54	from chronic myelogenous leukemia	bone marrow			pDNR-LIB (Clontech)
L2667	NT0013		nervous_tumor			puc18
L2669	NT0022		nervous_tumor			puc18
L2670	NT0023		nervous_tumor			puc18
L2686	NT0058		nervous_tumor			puc18
L2702	NT0098		nervous_tumor			puc18
L2709	NT0105		nervous_tumor			puc18
L2799	FT0096		prostate_tumor			puc18
L2804	FT0103		prostate_tumor			puc18
L2817	FT0131		prostate_tumor			puc18
L2831	FT0162		prostate_tumor			puc 18
L2842	0000MN		uterus			puc18
L2854	1600MN		uterus			puc18
L2884	AN0041		amnion_normal			puc18
L2904	BN0042		breast_normal			puc18
L2906	BN0047		breast_normal			puc18
L2910	BN0070		breast_normal			puc18
L2962	BN0221		breast_normal			puc18
L3002	BN0276		breast_normal			puc18
L3019	BN0303		breast_normal			puc18
L3071	EN0026		lung_normal			puc18
L3081	ET0005		lung_tumor	-		puc18
L3089	ET0018		lung_tumor			puc18
L3092	ET0023		lung_tumor			puc18
L3104	ET0041		lung_tumor			puc18
L3127	ET0084		lung_tumor			puc18
L3140	MT0031		marrow			puc18
L3153	MT0049		татом			puc18
L3154	MT0050		marrow			puc18

Code	Description	Ticane	Organ	Cell Line	Dispase	Vector
+-	OT0019		ovary			puc18
L3210	OT0067		ovary			puc18
L3212	OT0076		ovary			puc18
L3215	OT0083		ovary			puc18
L3226	FN0019		prostate_normal			puc18
L3255	FN0064		prostate_normal			puc18
L3262	FN0073		prostate_normal			puc18
L3316	FN0188		prostate_normal			puc18
L3352	TN0027		testis_normal			puc18
L3374	TN0070		testis_normal			puc18
┝	TN0079		testis_normal			puc18
L3387	GKB	hepatocellular carcinoma				pBluescript sk(-)
L3388	GKC	hepatocellular carcinoma				pBluescript sk(-)
L3391	NIH_MGC_53	carcinoma, cell line	bladder			pDNR-LIB (Clontech)
L3402	AN0086		amnion_normal			puc18
L3403	AN0087		amnion_normal			puc18
L3404	AN0089		amnion_normal			puc18 .
	BT0634		breast			puc18
L3459	FT0175		prostate_tumor			puc18
-	GN0057		placenta_normal			puc18
L3484	GN0067		placenta_normal			puc18
L3485	GN0070		placenta_normal			puc18
L3491	GN0076		placenta_normal			puc18
L3499	HT0617		head_neck			puc18
L3504	HT0873		head_neck			puc18
L3506	HT0879		head_neck			puc18
L3521	HT0919		head_neck			puc18
L3530	HT0939		head_neck			puc18
L3603	UM0093		uterus			puc18
L3612	UT0011		uterus_tumor			puc18
L3618	UT0050		uterus_tumor			puc18

Code	Description	Tissue	Organ	Cell Line	Disease	Vector
L3636	NIH_MGC_73		brain			pDNR-LIB (Clontech)
L3642	ADA	Adrenal gland				pBluescript sk(-)
L3643	ADB	Adrenal gland				pBluescript sk(-)
L3644	ADC	Adrenal gland				pBluescript sk(-)
L3645	r _O	adrenal cortico adenoma for Cushing"s syndrome				pBluescript sk(-)
L3646	DCA					pTriplEx2
L3649	DCB	•				pTriplEx2
L3653	HTB	Hypothalamus				pBluescript sk(-)
L3655	HTC	Hypothalamus				pBluescript sk(-)
L3657	HTF	Hypothalamus				pBluescript sk(-)
L3658	cdA	pheochromocytoma				pTriplEx2
L3659	CB	cord blood				pBluescript
L3661	NPA	pituitary				pBluescript sk(-)
L3665	NIH_MGC_75		kidney			pDNR-LIB (Clontech)
L3673	AN0084		amnion_normal			puc18
_	CT0524		colon			puc18
L3808	UT0078		uterus_tumor			puc18
L3811	NPC	pituitary				pBluescript sk(-)
L3812	NPD	pituitary				pBluescript sk(-)
L3813	TP	pituitary tumor				pTriplEx2
L3814	BM	Bone marrow				pTriplEx2
L3815	MDS	Bone marrow				pTriplEx2
L3816	HEMBA1	whole embryo, mainly head				pME18SFL3
L3817	HEMBB1	whole embryo, mainly body				pME18SFL3
L3819	NIH_MGC_76		liver			pDNR-LIB (Clontech)
L3823	NT2RM1			NT2		pUC19FL3
L3824	NT2RM2			NT2		pME18SFL3
L3825	NT2RM4			NT2		pME18SFL3
L3827	NT2RP2			NT2		pME18SFL3
L3828	NT2RP3			NT2		pME18SFL3

Code	Description	Tissua	Organ	Cell Line	Disease	Vector
L3829	NT2RP4			NT2		pME18SFL3
L3831	OVARCI	ovary, tumor tissue				pME18SFL3
L3832	PLACE1	placenta				pME18SFL3
L3833	PLACE2	placenta				pME18SFL3
L3834	PLACE3	placenta		•		pME18SFL3
L3837	THYROI	thyroid gland				pME18SFL3
L3841	NIH_MGC_18	large cell carcinoma	gunl			pOTB7
L3872	NCI_CGAP_Skn1		skin, normal, 4 pooled sa			pCMV-SPORT6
L3904	NCI_CGAP_Bm64	glioblastoma with EGFR amplification	brain			pCMV-SPORT6
L3905	NCI_CGAP_Bm67	anaplastic oligodendroglioma with 1p/19q loss	brain			pCMV-SPORT6
L4497	NCI_CGAP_Br22	invasive ductal carcinoma, 3 pooled samples	breast			pCMV-SPORT6
1.4501	NCI_CGAP_Sub8					pT7T3D-Pac (Pharmacia) with a modified polylinker
1.4556	NCI_CGAP_HN13	squamous cell carcinoma	tongue			pCMV-SPORT6
1.4558	NCI_CGAP_Pan3		pancreas			pCMV-SPORT6
1,4669	NCI_CGAP_0v41	serous papillary tumor	ovary			pCMV-SPORT6
L4747	NCI_CGAP_Bm41	oligodendroglioma	brain			pT7T3D-Pac (Pharmacia) with a modified polylinker
L5286	NCI_CGAP_Thy10	medullary carcinoma	thyroid			pAMP10
L5565	NCI_CGAP_Bm66	glioblastoma with probably TP53 mutation and witho	brain			pCMV-SPORT6
L5566	NCI_CGAP_Bm70	anaplastic oligodendroglioma	brain			pCMV-SPORT6.ccdb
L5574	-	normal epithelium	nasopharynx			pAMP10
L5575	NCI_CGAP_Bm65	glioblastoma without EGFR amplification	brain			pCMV-SPORT6
L5622	NCI_CGAP_Skn3		skin		•	pCMV-SPORT6
L5623		squamous cell carcinoma	skin			pCMV-SPORT6

## **Description of Table 5**

Table 5 provides a key to the OMIM reference identification numbers disclosed in Table 1B.1, column 9. OMIM reference identification numbers (Column 1) were derived from Online Mendelian Inheritance in Man (Online Mendelian Inheritance in Man, OMIM. McKusick-Nathans Institute for Genetic Medicine, Johns Hopkins University (Baltimore, MD) and National Center for Biotechnology Information, National Library of Medicine, (Bethesda, MD) 2000. World Wide Web URL: http://www.ncbi.nlm.nih.gov/omim/). Column 2 provides diseases associated with the cytologic band disclosed in Table 1B.1, column 8, as determined using the Morbid Map database.

## TABLE 5

OMIM Reference	Description
100690	Myasthenic syndrome, slow-channel congenital, 601462
100710	Myasthenic syndrome, slow-channel congenital, 601462
101000	Meningioma, NF2-related, sporadic Schwannoma, sporadic
101000	Neurofibromatosis, type 2
101000	Neurolemmomatosis
101000	Malignant mesothelioma, sporadic
102200	Somatotrophinoma
102772	[AMP deaminase deficiency, erythrocytic]
103581	Albright hereditary osteodystrophy-2
103600	[Dysalbuminemic hyperthyroxinemia]
103600	[Dysalbuminemic hyperzincemia], 194470
103600	Analbuminemia
103850	Aldolase A deficiency
104150	[AFP deficiency, congenital]
104150	[Hereditary persistence of alpha-fetoprotein]
104500	Amelogenesis imperfecta-2, hypoplastic local type
104770	Amyloidosis, secondary, susceptibility to
106100	Angioedema, hereditary
106150	Hypertension, essential, susceptibility to
106150	Preeclampsia, susceptibility to
106180	Myocardial infarction, susceptibility to
106210	Peters anomaly
106210	Cataract, congenital, with late-onset corneal dystrophy
106210	Foveal hypoplasia, isolated, 136520
106210	Aniridia
107271	CD59 deficiency
107300	Antithrombin III deficiency
107670	Apolipoprotein A-II deficiency
107741	Hyperlipoproteinemia, type III
107777	Diabetes insipidus, nephrogenic, autosomal recessive, 222000
109270	Renal tubular acidosis, distal, 179800
109270	Spherocytosis, hereditary
109270	[Acanthocytosis, one form]
109270	[Elliptocytosis, Malaysian-Melanesian type]

OMIM Reference	Description
109270	Hemolytic anemia due to band 3 defect
109400	Basal cell nevus syndrome
110700	Vivax malaria, susceptibility to
112261	Fibrodysplasia ossificans progressiva
113100	Brachydactyly, type C
113900	Heart block, progressive familial, type I
114550	Hepatocellular carcinoma
114835	Monocyte carboxyesterase deficiency
115500	Acatalasemia
115665	Cataract, congenital, Volkmann type
116800	Cataract, Marner type
116806	Colorectal cancer
116860	Cavernous angiomatous malformations
118485	Polycystic ovary syndrome with hyperandrogenemia
118800	Choreoathetosis, familial paroxysmal
120070	Alport syndrome, autosomal recessive, 203780
120120	Epidermolysis bullosa dystrophica, dominant, 131750
120120	Epidermolysis bullosa dystrophica, recessive, 226600
120120	Epidermolysis bullosa, pretibial, 131850
120131	Alport syndrome, autosomal recessive, 203780
120131	Hematuria, familial benign
120140	Osteoarthrosis, precocious
120140	SED congenita
120140	SMED Strudwick type
120140	Stickler syndrome, type I
120140	Wagner syndrome, type II
120140	Achondrogenesis-hypochondrogenesis, type II
120140	Kniest dysplasia
120150	Osteogenesis imperfecta, 4 clinical forms, 166200, 166210, 259420, 166220
120150	Osteoporosis, idiopathic, 166710
120150	Ehlers-Danlos syndrome, type VIIA1, 130060
120215	Ehlers-Danlos syndrome, type I, 130000
120215	Ehlers-Danlos syndrome, type II, 130010
120220	Bethlem myopathy, 158810
120240	Bethlem myopathy, 158810
120260	Epiphyseal dysplasia, multiple, type 2, 600204
120435	Muir-Torre syndrome, 158320
120435	Colorectal cancer, hereditary, nonpolyposis, type 1 Ovarian cancer
120436	Muir-Torre family cancer syndrome, 158320
120436	Turcot syndrome with glioblastoma, 276300
120436	Colorectal cancer, hereditary nonpolyposis, type 2
120550	Cladeficiency, type A
120570	C1q deficiency, type B
120575	C1q deficiency, type C
120950	C8 deficiency, type I
120960	C8 deficiency, type II
121050	Contractural arachnodactyly, congenital
121800	Corneal dystrophy, crystalline, Schnyder
122720	Nicotine addiction, protection from
122720	Coumarin resistance, 122700
123000	Craniometaphyseal dysplasia

OMIM Reference	Description
123270	[Creatine kinase, brain type, ectopic expression of]
123580	Cataract, congenital, autosomal dominant
123620	Cataract, cerulean, type 2, 601547
123660	Cataract, Coppock-like
123940	White sponge nevus, 193900
124200	Darier disease (keratosis follicularis)
125370	Dentatorubro-pallidoluysian atrophy
125660	Myopathy, desminopathic
125660	Cardiomyopathy
126060	Anemia, megaloblastic, due to DHFR deficiency
126090	Hyperphenylalaninemia due to pterin-4a-carbinolamine dehydratase
120070	deficiency, 264070
126337	Myxoid liposarcoma
126340	Xeroderma pigmentosum, group D, 278730
126391	DNA ligase I deficiency
126600	Doyne honeycomb retinal dystrophy
126600	Drusen, radial, autosomal dominant
129010	Neuropathy, congenital hypomyelinating, 1
129900	EEC syndrome-1
130410	Glutaricaciduria, type IIB
130500	Elliptocytosis-1
131100	Multiple endocrine neoplasia I
131100	Prolactinoma, hyperparathyroidism, carcinoid syndrome
131100	Carcinoid tumor of lung
131210	Atherosclerosis, susceptibility to
	Eosinophilia, familial
131400 132800	Basal cell carcinoma
	Epithelioma, self-healing, squamous 1, Ferguson-Smith type
132800 133200	Erythrokeratodermia variabilis
133701	Exostoses, multiple, type 2  Vitreoretinopathy, exudative, familial
133780	Hyperferritinemia-cataract syndrome, 600886
134790	
134934	Thanatophoric dysplasia, types I and II, 187600 Achondroplasia, 100800
134934	
134934	Craniosynostosis, nonsyndromic Crouzon syndrome with acanthosis nigricans
134934	
134934	Hypochondroplasia, 146000
135300	Fibromatosis, gingival
135940	Ichthyosis vulgaris, 146700
136132	[Fish-odor syndrome], 602079
136435	Ovarian dysgenesis, hypergonadotropic, with normal karyotype, 233300
136530	Male infertility, familial
138030	[Hyperproglucagonemia]
138040	Cortisol resistance
138140	Glucose transport defect, blood-brain barrier
138300	Hemolytic anemia due to glutathione reductase deficiency
138320	Hemolytic anemia due to glutathione peroxidase deficiency
138570	Non-insulin dependent diabetes mellitus, susceptibility to
138700	[Apolipoprotein H deficiency]
138760	[Glyoxalase II deficiency]
138981	Pulmonary alveolar proteinosis, 265120

OMIM Reference	Description
139190	Gigantism due to GHRF hypersecretion
139190	Isolated growth hormone deficiency due to defect in GHRF
139250	Isolated growth hormone deficiency, Illig type with absent GH and Kowarski
	type with bioinactive GH
139350	Epidermolytic hyperkeratosis, 113800
139350	Keratoderma, palmoplantar, nonepidermolytic
140100	[Anhaptoglobinemia]
140100	[Hypohaptogloginemia]
141750	Alpha-thalassemia/mental retardation syndrome, type 1
141800	Methemoglobinemias, alpha-
141800	Thalassemias, alpha-
141800	Erythremias, alpha-
141800	Heinz body anemias, alpha-
141850	Thalassemia, alpha-
141850	Erythrocytosis
141850	Heinz body anemia
141850	Hemoglobin H disease
141850	Hypochromic microcytic anemia
142600	Hemolytic anemia due to hexokinase deficiency
142989	Synpolydactyly, type II, 186000
143100	Huntington disease
143200	Wagner syndrome
143200	Erosive vitreoretinopathy
143890	Hypercholesterolemia, familial
144120	Hyperimmunoglobulin G1 syndrome
145001	Hyperparathyroidism-jaw tumor syndrome
145260	Pseudohypoaldosteronism, type II
146150	Hypomelanosis of Ito
146760	[IgG receptor I, phagocytic, familial deficiency of]
146790	Lupus nephritis, susceptibility to
147020	Agammaglobulinemia, 601495
147050	Atopy
147110	IgG2 deficiency, selective
147440	Growth retardation with deafness and mental retardation
148040	Epidermolysis bullosa simplex, Koebner, Dowling-Meara, and Weber-
140040	Cockayne types, 131900, 131760, 131800
148041	Pachyonychia congenita, Jadassohn-Lewandowsky type, 167200
148043	Meesmann corneal dystrophy, 122100
148065	White sponge nevus, 193900
148070	Liver disease, susceptibility to, from hepatotoxins or viruses
148080	Epidermolytic hyperkeratosis, 113800
148900	Klippel-Feil syndrome with laryngeal malformation
150200	[Placental lactogen deficiency]
151385	Leukemia, acute myeloid
151390	Leukemia, acute T-cell
151410	Leukemia, chronic myeloid
151410	Leukemia, T-cell acute lymphoblastoid
151440	Hepatic lipase deficiency
	Coronary artery disease, susceptibility to
152200 152445	Vohwinkel syndrome, 124500
	<del></del>
152445	Erythrokeratoderma, progressive symmetric, 602036

OMIM Reference	Description
152790	Precocious puberty, male, 176410
152790	Leydig cell hypoplasia
153454	Ehlers-Danlos syndrome, type VI, 225400
153455	Cutis laxa, recessive, type I, 219100
153700	Macular dystrophy, vitelliform type
154275	Malignant hyperthermia susceptibility 2
154400	Acrofacial dysostosis, Nager type
154545	Chronic infections, due to opsonin defect
155555	[Red hair/fair skin]
155555	UV-induced skin damage, vulnerability to
156232	Mesomelic dysplasia, Kantaputra type
156850	Cataract, congenital, with microphthalmia
157147	Abetalipoproteinemia, 200100
157170	Holoprosencephaly-2
157640	PEO with mitochondrial DNA deletions, type 1
158590	Spinal muscular atrophy-4
159000	Muscular dystrophy, limb-girdle, type 1A
159001	Muscular dystrophy, limb-girdle, type 1B
160781	Cardiomyopathy, hypertrophic, mid-left ventricular chamber type
160900	Myotonic dystrophy
160980	Carney myxoma-endocrine complex
161015	Mitochondrial complex I deficiency, 252010
162150	Obestiy with impaired prohormone processing, 600955
163950	Noonan syndrome-1
163950	Cardiofaciocutaneous syndrome, 115150
164009	Leukemia, acute promyelocytic, NUMA/RARA type
164500	Spinocerebellar ataxia-7
164731	Ovarian carcinoma, 167000
164920	Piebaldism
164920	Mast cell leukemia
164920	Mastocytosis with associated hematologic disorder
167000	Ovarian cancer, serous
167410	Rhabdomyosarcoma, alveolar, 268220
168360	Paraneoplastic sensory neuropathy
168461	Multiple myeloma, 254250
168461	Parathyroid adenomatosis 1
168461	Centrocytic lymphoma
168468	Metaphyseal chondrodysplasia, Murk Jansen type, 156400
168500	Parietal foramina
170650	Periodontitis, juvenile
171190	Hypertension, essential, 145500
171650	Lysosomal acid phosphatase deficiency
171760	Hypophosphatasia, adult, 146300
171760	Hypophosphatasia, infantile, 241500
171860	Hemolytic anemia due to phosphofructokinase deficiency
172430	Enolase deficiency
173610	Platelet alpha/delta storage pool deficiency
173850	Polio, susceptibility to
173870	
117010	Xeroderma pigmentosum
173870	Fanconi anemia

OMIM Reference	Description
174810	Osteolysis, familial expansile
174900	Polyposis, juvenile intestinal
176100	Porphyria cutanea tarda
176100	Porphyria, hepatoerythropoietic
176261	Jervell and Lange-Nielsen syndrome, 220400
176270	Prader-Willi syndrome
176640	Creutzfeldt-Jakob disease, 123400
176640	Gerstmann-Straussler disease, 137440
176640	Insomnia, fatal familial
176880	Protein S deficiency
176930	Dysprothrombinemia
176930	Hypoprothrombinemia
176960	Pituitary tumor, invasive
178300	Ptosis, hereditary congenital, 1
178600	Pulmonary hypertension, familial primary
179095	Male infertility
179615	Reticulosis, familial histiocytic, 267700
179615	Severe combined immunodeficiency, B cell-negative, 601457
179616	Severe combined immunodeficiency, B cell-negative, 601457
179755	Renal cell carcinoma, papillary, 1
180071	Retinitis pigmentosa, autosomal recessive
180072	Night blindness, congenital stationary, type 3, 163500
180072	Retinitis pigmentosa, autosomal recessive
180105	Retinitis pigmentosa-10
180200	Osteosarcoma, 259500
180200	Pinealoma with bilateral retinoblastoma
180200	Retinoblastoma
180200	Bladder cancer, 109800
180385	Leukemia, acute T-cell
180721	Retinitis pigmentosa, digenic
180840	Susceptibility to IDDM
181430	Scapuloperoneal syndrome, myopathic type
181460	Schistosoma mansoni, susceptibility/resistance to
181510	Schizophrenia
182280	Small-cell cancer of lung
182600	Spastic paraplegia-3A
182601	Spastic paraplegia-4
182860	Pyropoikilocytosis
182860	Spherocytosis, recessive
182860	Elliptocytosis-2
185800	Symphalangism, proximal
186580	Arthrocutaneouveal granulomatosis
186855	Leukemia-2, T-cell acute lymphoblastic
186880	Leukemia/lymphoma, T-cell
187040	Leukemia-1, T-cell acute lymphoblastic
188826	Sorsby fundus dystrophy, 136900
189800	Preeclampsia/eclampsia
190195	Ichthyosiform erythroderma, congenital, 242100
190195	Ichthyosis, lamellar, autosomal recessive, 242300
190198	Leukemia, T-cell acute lymphoblastic
190685	Down syndrome

OMIM Reference	Description
191092	Tuberous sclerosis-2
191181	Cervical carcinoma
191315	Insensitivity to pain, congenital, with anhidrosis, 256800
192090	Ovarian carcinoma
192090	Breast cancer, lobular
192090	Endometrial carcinoma
192090	Gastric cancer, familial, 137215
192974	Neonatal alloimmune thrombocytopenia
192974	Glycoprotein Ia deficiency
193235	Vitreoretinopathy, neovascular inflammatory
193300	Renal cell carcinoma
193300	von Hippel-Lindau syndrome
193500	Rhabdomyosarcoma, alveolar, 268220
193500	Waardenburg syndrome, type I
193500	Waardenburg syndrome, type III, 148820
193500	Craniofacial-deafness-hand syndrome, 122880
194070	Wilms tumor, type 1
194070	Denys-Drash syndrome
194070	Frasier syndrome, 136680
194190	Wolf-Hirschhorn syndrome
201460	Acyl-CoA dehydrogenase, long chain, deficiency of
205100	Amyotrophic lateral sclerosis, juvenile
207750	Hyperlipoproteinemia, type Ib
208400	Aspartylglucosaminuria
209901	Bardet-Biedl syndrome 1
212138	Carnitine-acylcarnitine translocase deficiency
216550	Cohen syndrome
218000	Andermann syndrome
221820	Gliosis, familial progressive subcortical
222700	Lysinuric protein intolerance
222800	Hemolytic anemia due to bisphosphoglycerate mutase deficiency
222900	Sucrose intolerance
223900	Dysautonomia, familial
224100	Congenital dyserythropoietic anemia II
225500	Ellis-van Creveld syndrome
227220	[Eye color, brown]
227646	Fanconi anemia, type D
227650	Fanconi anemia, type A
230000	Fucosidosis
230400	Galactosemia
230800	Gaucher disease
230800	Gaucher disease with cardiovascular calcification
231550	Achalasia-addisonianism-alacrimia syndrome
231675	Glutaricaciduria, type IIC
231680	Glutaricaciduria, type IIA
232500	Glycogen storage disease IV
232600	McArdle disease
232800	Glycogen storage disease VII
233700	Chronic granulomatous disease due to deficiency of NCF-1
236100	Holoprosencephaly-1
236200	Homocystinuria, B6-responsive and nonresponsive types

OMIM Reference	Description
236250	Homocystinuria due to MTHFR deficiency
236700	McKusick-Kaufman syndrome
236730	Urofacial syndrome
237300	Carbamoylphosphate synthetase I deficiency
238310	Hyperglycinemia, nonketotic, type II
240300	Autoimmune polyglandular disease, type I
240400	Scurvy
245200	Krabbe disease
245349	Lacticacidemia due to PDX1 deficiency
	Norum disease
245900	Fish-eye disease
245900	HMG-CoA lyase deficiency
246450	Mannosidosis, beta-
248510	
248600	Maple syrup urine disease, type Ia
249000	Meckel syndrome
249100	Familial Mediterranean fever
250250	Cartilage-hair hypoplasia
250790	Methemoglobinemia due to cytochrome b5 deficiency
250850	Hypermethioninemia, persistent, autosomal dominant, due to methionine
	adenosyltransferase I/III deficiency
251170	Mevalonicaciduria
251600	Microphthalmia, autosomal recessive
252800	Mucopolysaccharidosis Ih
252800	Mucopolysaccharidosis Ih/s
252800	Mucopolysaccharidosis Is
253000	Mucopolysaccharidosis IVA
253200	Maroteaux-Lamy syndrome, several forms
253250	Mulibrey nanism
253800	Walker-Warburg syndrome, 236670
253800	Fukuyama type congenital muscular dystrophy
255800	Schwartz-Jampel syndrome
256700	Neuroblastoma
258501	3-methylglutaconicaciduria, type III
259700	Osteopetrosis, recessive
259770	Osteoporosis-pseudoglioma syndrome
259900	Hyperoxaluria, primary, type 1
262000	Bjornstad syndrome
266200 .	Anemia, hemolytic, due to PK deficiency
266600	Inflammatory bowel disease-1
267750	Knobloch syndrome
268800	Sandhoff disease, infantile, juvenile, and adult forms
268800	Spinal muscular atrophy, HEXB-related
270100	Situs inversus viscerum
272750	GM2-gangliosidosis, AB variant
272800	Tay-Sachs disease
272800	[Hex A pseudodeficiency]
272800	GM2-gangliosidosis, juvenile, adult
274180	Thromboxane synthase deficiency
276600	Tyrosinemia, type II
276700	Tyrosinemia, type I
276710	Tyrosinemia, type III
2/0/10	1 1 Jacontonia, type in

OMIM Reference	Description
276900	Usher syndrome, type 1A
276901	Usher syndrome, type 2
278700	Xeroderma pigmentosum, group A
300011	Menkes disease, 309400
300011	Occipital horn syndrome, 304150
300011	Cutis laxa, neonatal
300046	Mental retardation, X-linked 23, nonspecific
300047	Mental retardation, X-linked 20
300067	Subcortical laminar heterotopia, X-linked dominant
300067	Lissencephaly, X-linked
300071	Night blindness, congenital stationary, type 2
300075	Coffin-Lowry syndrome, 303600
300077	Mental retardation, X-linked 29
300110	Night blindness, congenital stationary, X-linked incomplete, 300071
300121	Subcortical laminal heteropia, X-linked, 300067
300121	Lissencephaly, X-linked, 300067
300127	Mental retardation, X-linked, 60
300600	Ocular albinism, Forsius-Eriksson type
301000	Thrombocytopenia, X-linked, 313900
301000	Wiskott-Aldrich syndrome
301200	Amelogenesis imperfecta
301201	Amelogenesis imperfecta-3, hypoplastic type
301830	Arthrogryposis, X-linked (spinal muscular atrophy, infantile, X-linked)
301835	Arts syndrome
302350	Nance-Horan syndrome
302801	Charcot-Marie-Tooth neuropathy, X-linked-2, recessive
305435	Heterocellular hereditary persistence of fetal hemoglobin, Swiss type
305450	FG syndrome
306000	Glycogenosis, X-linked hepatic, type I
306000	Glycogenosis, X-linked hepatic, type II
307800	Hypophosphatemia, hereditary
308800	Keratosis follicularis spinulosa decalvans
309470	Mental retardation, X-linked, syndromic-3, with spastic diplegia
309500	Renpenning syndrome-1
309510	Mental retardation, X-linked, syndromic-1, with dystonic movements, ataxia, and seizures
309605	Mental retardation, X-linked, syndromic-4, with congenital contractures and
200610	low fingertip arches  Mental retardation, X-linked, syndromic-2, with dysmorphism and cerebral
309610	•
200950	atrophy  Response condenses
309850 311050	Brunner syndrome Ontic attachy V linked
	Optic atrophy, X-linked
311200	Oral-facial-digital syndrome 1  Phosphoribosyl pyrophosphate synthetase-related gout
311850	<del> </del>
312040	N syndrome, 310465
312060	Properdin deficiency, X-linked
312170	Pyruvate dehydrogenase deficiency
312700	Retinoschisis
313400	Spondyloepiphyseal dysplasia tarda
313700	Perineal hypospadias
313700	Prostate cancer ·

OMIM Reference	Description
313700	Spinal and bulbar muscular atrophy of Kennedy, 313200
313700	Breast cancer, male, with Reifenstein syndrome
313700	Androgen insensitivity, several forms
314580	Wieacker-Wolff syndrome
600040	Colorectal cancer
600045	Xeroderma pigmentosum, group E, subtype 2
600065	Leukocyte adhesion deficiency, 116920
600079	Colon cancer
600101	Deafness, autosomal dominant 2
600119	Muscular dystrophy, Duchenne-like, type 2
600119	Adhalinopathy, primary
600140	Rubenstein-Taybi syndrome, 180849
600151	Bardet-Biedl syndrome 3
600163	Long QT syndrome-3
600175	Spinal muscular atrophy, congenital nonprogressive, of lower limbs
600194	Ichthyosis bullosa of Siemens, 146800
600223	Spinocerebellar ataxia-4
600231	Palmoplantar keratoderma, Bothnia type
600243	Temperature-sensitive apoptosis
600258	Colorectal cancer, hereditary nonpolyposis, type 3
600259	Turcot syndrome with glioblastoma, 276300
600259	Colorectal cancer, hereditary nonpolyposis, type 4
600266	Resistance/susceptibility to TB, etc.
600273	Polycystic kidney disease, infantile severe, with tuberous sclerosis
600276	Cerebral arteriopathy with subcortical infarcts and leukoencephalopathy,
000270	125310
600319	Diabetes mellitus, insulin-dependent, 4
600320	Insulin-dependent diabetes mellitus-5
600332	Rippling muscle disease-1
600354	Spinal muscular atrophy-1, 253300
600354	Spinal muscular atrophy-2, 253550
600354	Spinal muscular atrophy-3, 253400
600359	Bartter syndrome, type 2
600374	Bardet-Biedl syndrome 4
600512	Epilepsy, partial
600525	Trichodontoosseous syndrome, 190320
600528	CPT deficiency, hepatic, type I, 255120
600536	Myopathy, congenital
600593	Craniosynostosis, Adelaide type
600623	Prostate cancer, 176807
600631	Enuresis, nocturnal, 1
600650	Myopathy due to CPT II deficiency, 255110
600650	CPT deficiency, hepatic, type II, 600649
600678	Cancer susceptibility
600698	Salivary adenoma
600698	Uterine leiomyoma
600698	Lipoma
600698	Liponatosis, mutiple, 151900
600722	Ceroid lipofuscinosis, neuronal, variant juvenile type, with granular
	osmiophilic deposits
600722	Ceroid lipofuscinosis, neuronal-1, infantile, 256730

OMIM Reference	Description
600759	Alzheimer disease-4
600760	Pseudohypoaldosteronism, type I, 264350
600760	Liddle syndrome, 177200
600761	Pseudohypoaldosteronism, type I, 264350
600761	Liddle syndrome, 177200
600795	Dementia, familial, nonspecific
600807	Bronchial asthma
600808	Enuresis, nocturnal, 2
600811	Xeroderma pigmentosum, group E, DDB-negative subtype, 278740
600850	Schizophrenia disorder-4
600852	Retinitis pigmentosa-17
600882	Charcot-Marie-Tooth neuropathy-2B
600883	Diabetes mellitus, insulin-dependent, 8
600887	Endometrial carcinoma
600897	Cataract, zonular pulverulent-1, 116200
600900	Muscular dystrophy, limb-girdle, type 2E
600956	Persistent Mullerian duct syndrome, type II, 261550
600958	Cardiomyopathy, familial hypertrophic, 4, 115197
600965	Deafness, autosomal dominant 6
600975	Glaucoma 3, primary infantile, B
600996	Arrhythmogenic right ventricular dysplasia-2
601002	5-oxoprolinuria, 266130
601002	Hemolytic anemia due to glutathione synthetase deficiency, 231900
601072	Deafness, autosomal recessive 8
601072	Pycnodysostosis, 265800
601145	Epilepsy, progressive myoclonic 1, 254800
601146	Brachydactyly, type C, 113100
601146	Acromesomelic dysplasia, Hunter-Thompson type, 201250
601146	Chondrodysplasia, Grebe type, 200700
601226	Progressive external ophthalmoplegia, type 2
601277	Ichthyosis, lamellar, type 2
601277	Hereditary hemorrhagic telangiectasia-2, 600376
601313	Polycystic kidney disease, adult type I, 173900
601362	DiGeorge syndrome/velocardiofacial syndrome complex-2
601386	Deafness, autosomal recessive 12
601399	Platelet disorder, familial, with associated myeloid malignancy
601402	Leukemia, myeloid, acute
601412	Deafness, autosomal dominant 7
601412	Inflammatory bowel disease-2
601493	Cardiomyopathy, dilated 1C
601517	Spinocerebellar ataxia-2, 183090
601518	Prostate cancer, hereditary, 1, 176807
601567	Combined factor V and VIII deficiency, 227300
601596	Charcot-Marie-Tooth neuropathy, demyelinating
601623	Angelman syndrome
601652	Glaucoma 1A, primary open angle, juvenile-onset, 137750
	Hirschsprung disease, one form
601669	
601692	Reis-Bucklers corneal dystrophy
601692	Corneal dystrophy, Avellino type
601692	Corneal dystrophy, Groenouw type I, 121900
601692	Corneal dystrophy, lattice type I, 122200

OMIM Reference	Description
601744	Systemic lupus erythematosus, susceptibility to, 1
601769	Osteoporosis, involutional
601769	Rickets, vitamin D-resistant, 277440
601771	Glaucoma 3A, primary infantile, 231300
601780	Ceroid-lipofuscinosis, neuronal-6, variant late infantile
601785	Carbohydrate-deficient glycoprotein syndrome, type I, 212065
601800	[Hair color, brown]
601843	Hypothyroidism, congenital, 274400
601844	Pseudohypoaldosteronism type II
601863	Bare lymphocyte syndrome, complementation group C
601884	[High bone mass]
601889	Lymphoma, diffuse large cell
601920	Alagille syndrome, 118450
601928	Monilethrix, 158000
601975	Ectodermal dysplasia/skin fragility syndrome
602080	Paget disease of bone-2
602088	Nephronophthisis, infantile
602089	Hemangioma, capillary, hereditary
602092	Deafness, autosomal recessive 18
602116	Glioma
602117	Prader-Willi syndrome
602121	Deafness, autosomal dominant nonsyndromic sensorineural, 1, 124900
602153	Monilethrix, 158000
602225	Cone-rod retinal dystrophy-2, 120970
602225	Leber congenital amaurosis, type III
602279	Oculopharyngeal muscular dystorphy, 164300
602279	Oculopharyngeal muscular dystrophy, autosomal recessive, 257950
602363	Ellis-van Creveld-like syndrome
602460	Deafness, autosomal dominant 15, 602459
602491	Hyperlipidemia, familial combined, 1
602544	Parkinson disease, juvenile, type 2, 600116
602568	Homocystinuria-megaloblastic anemia, cbl E type, 236270
602574	Deafness, autosomal dominant 12, 601842
602574	Deafness, autosomal dominant 8, 601543
602629	Dystonia-6, torsion
602783	Spastic paraplegia-7

## Mature Polypeptides

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The present invention also encompasses mature forms of a polypeptide having the amino acid sequence of SEQ ID NO:Y and/or the amino acid sequence encoded by the cDNA in a deposited clone. Polynucleotides encoding the mature forms (such as, for example, the polynucleotide sequence in SEQ ID NO:X and/or the polynucleotide sequence contained in the cDNA of a deposited clone) are also encompassed by the invention. Moreover, fragments or variants of these polypeptides (such as, fragments as described herein, polypeptides at least 80%, 85%, 90%, 95%, 96%, 97%, 98%, 99%, or 100% identical to these polypeptides, or polypeptides

encoded by a polynucleotide that hybridizes under stringent conditions to the complementary strand of the polynucleotide encoding these polypeptides) are also encompassed by the invention. In preferred embodiments, these fragments or variants retain one or more functional activities of the full-length or mature form of the polypeptide (e.g., biological activity (such as, for example, activity useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders), antigenicity (ability to bind, or compete with a polypeptide of the invention for binding, to an anti-polypeptide of the invention antibody), immunogenicity (ability to generate antibody which binds to a specific polypeptide of the invention), ability to form multimers with polypeptides of the invention, and ability to bind to a receptor or ligand for a polypeptide of the invention). Antibodies that bind the polypeptides of the invention, and polynucleotides encoding these polypeptides are also encompassed by the invention.

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According to the signal hypothesis, proteins secreted by mammalian cells have a signal or secretary leader sequence which is cleaved from the mature protein once export of the growing protein chain across the rough endoplasmic reticulum has been initiated. Most mammalian cells and even insect cells cleave secreted proteins with the same specificity. However, in some cases, cleavage of a secreted protein is not entirely uniform, which results in two or more mature species of the protein. Further, it has long been known that cleavage specificity of a secreted protein is ultimately determined by the primary structure of the complete protein, that is, it is inherent in the amino acid sequence of the polypeptide.

Methods for predicting whether a protein has a signal sequence, as well as the cleavage point for that sequence, are available. For instance, the method of McGeoch, Virus Res. 3:271-286 (1985), uses the information from a short N-terminal charged region and a subsequent uncharged region of the complete (uncleaved) protein. The method of von Heinje, Nucleic Acids Res. 14:4683-4690 (1986) uses the information from the residues surrounding the cleavage site, typically residues -13 to +2, where +1 indicates the amino terminus of the secreted protein. The accuracy of predicting the cleavage points of known mammalian secretory proteins for each of these methods is in the range of 75-80%. (von Heinje, supra.) However, the two methods do not always produce the same predicted cleavage point(s) for a given protein.

In the present case, the deduced amino acid sequence of the secreted polypeptide was analyzed by a computer program called SignalP (Henrik Nielsen et al., Protein Engineering 10:1-6 (1997)), which predicts the cellular location of a protein based on the amino acid sequence. As part of this computational prediction of localization, the methods of McGeoch and von Heinje are incorporated. The analysis of the amino acid sequences of the secreted proteins described herein by this program provided the results shown in Table 1A.

In specific embodiments, polypeptides of the invention comprise, or alternatively consist of, the predicted mature form of the polypeptide as delineated in columns 14 and 15 of Table 1A.

Moreover, fragments or variants of these polypeptides (such as, fragments as described herein, polypeptides at least 80%, 85%, 90%, 95%, 96%, 97%, 98%, 99%, or 100% identical to these polypeptides, or polypeptides encoded by a polynucleotide that hybridizes under stringent conditions to the complementary strand of the polynucleotide encoding these polypeptides) are also encompassed by the invention. In preferred embodiments, these fragments or variants retain one or more functional acitivities of the full-length or mature form of the polypeptide (e.g., biological activity (such as, for example, activity useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders), antigenicity (ability to bind, or compete with a polypeptide of the invention for binding, to an anti-polypeptide of the invention antibody), immunogenicity (ability to generate antibody which binds to a specific polypeptide of the invention), ability to form multimers with polypeptides of the invention, and ability to bind to a receptor or ligand for a polypeptide of the invention). Antibodies that bind the polypeptides of the invention, and polynucleotides encoding these polypeptides are also encompassed by the invention.

Polynucleotides encoding proteins comprising, or consisting of, the predicted mature form of polypeptides of the invention (e.g., polynucleotides having the sequence of SEQ ID NO: X (Table 1A, column 4), the sequence delineated in columns 7 and 8 of Table 1A, and a sequence encoding the mature polypeptide delineated in columns 14 and 15 of Table 1A (e.g., the sequence of SEQ ID NO:X encoding the mature polypeptide delineated in columns 14 and 15 of Table 1)) are also encompassed by the invention, as are fragments or variants of these polynucleotides (such as, fragments as described herein, polynucleotides at least 80%, 85%, 90%, 95%, 96%, 97%, 98%, 99%, or 100% identical to these polyncueotides, and nucleic acids which hybridizes under stringent conditions to the complementary strand of the polynucleotide).

As one of ordinary skill would appreciate, however, cleavage sites sometimes vary from organism to organism and cannot be predicted with absolute certainty. Accordingly, the present invention provides secreted polypeptides having a sequence shown in SEQ ID NO:Y which have an N-terminus beginning within 15 residues of the predicted cleavage point (i.e., having 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, or 15 more or less contiguous residues of SEQ ID NO:Y at the N-terminus when compared to the predicted mature form of the polypeptide (e.g., the mature polypeptide delineated in columns 14 and 15 of Table 1). Similarly, it is also recognized that in some cases, cleavage of the signal sequence from a secreted protein is not entirely uniform, resulting in more than one secreted species. These polypeptides, and the polynucleotides encoding such polypeptides, are contemplated by the present invention.

Moreover, the signal sequence identified by the above analysis may not necessarily predict the naturally occurring signal sequence. For example, the naturally occurring signal sequence may be further upstream from the predicted signal sequence. However, it is likely that the predicted signal sequence will be capable of directing the secreted protein to the ER.

Nonetheless, the present invention provides the mature protein produced by expression of the polynucleotide sequence of SEQ ID NO:X and/or the polynucleotide sequence contained in the cDNA of a deposited clone, in a mammalian cell (e.g., COS cells, as desribed below). These polypeptides, and the polynucleotides encoding such polypeptides, are contemplated by the present invention.

## Polynucleotide and Polypeptide Variants

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The present invention is also directed to variants of the polynucleotide sequence disclosed in SEQ ID NO:X or the complementary strand thereto, nucleotide sequences encoding the polypeptide of SEQ ID NO:Y, the nucleotide sequence of SEQ ID NO:X that encodes the polypeptide sequence as defined in columns 13 and 14 of Table 1A, nucleotide sequences encoding the polypeptide sequence as defined in columns 13 and 14 of Table 1A, the nucleotide sequence of SEQ ID NO:X encoding the polypeptide sequence as defined in Table 1B, nucleotide sequences encoding the polypeptide as defined in Table 1B, the nucleotide sequence as defined in columns 8 and 9 of Table 2, nucleotide sequences encoding the polypeptide encoded by the nucleotide sequence as defined in column 6 of Table 1C, nucleotide sequences encoding the polypeptide encoded by the nucleotide sequence as defined in column 6 of Table 1C, the cDNA sequence contained in ATCC Deposit No:Z, nucleotide sequences encoding the polypeptide encoded by the cDNA sequence contained in ATCC Deposit No:Z, and/or nucleotide sequences encoding a mature (secreted) polypeptide encoded by the cDNA sequence contained in ATCC Deposit No:Z.

The present invention also encompasses variants of the polypeptide sequence disclosed in SEQ ID NO:Y, the polypeptide as defined in columns 13 and 14 of Table 1A, the polypeptide sequence as defined in Table 1B, a polypeptide sequence encoded by the polynucleotide sequence in SEQ ID NO:X, a polypeptide sequence encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2, a polypeptide sequence encoded by the nucleotide sequence as defined in column 6 of Table 1C, a polypeptide sequence encoded by the complement of the polynucleotide sequence in SEQ ID NO:X, the polypeptide sequence encoded by the cDNA sequence contained in ATCC Deposit No:Z and/or a mature (secreted) polypeptide encoded by the cDNA sequence contained in ATCC Deposit No:Z.

"Variant" refers to a polynucleotide or polypeptide differing from the polynucleotide or polypeptide of the present invention, but retaining essential properties thereof. Generally, variants are overall closely similar, and, in many regions, identical to the polynucleotide or polypeptide of the present invention.

Thus, one aspect of the invention provides an isolated nucleic acid molecule comprising, or alternatively consisting of, a polynucleotide having a nucleotide sequence selected from the group consisting of: (a) a nucleotide sequence described in SEQ ID NO:X or contained in the

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cDNA sequence of ATCC Deposit No:Z; (b) a nucleotide sequence in SEQ ID NO:X or the cDNA in ATCC Deposit No:Z which encodes the complete amino acid sequence of SEQ ID NO:Y or the complete amino acid sequence encoded by the cDNA in ATCC Deposit No:Z; (c) a nucleotide sequence in SEQ ID NO:X or the cDNA in ATCC Deposit No:Z which encodes a mature polypeptide (i.e., a secreted polypeptide (e.g., as delineated in columns 14 and 15 of Table 1A)); (d) a nucleotide sequence in SEQ ID NO:X or the cDNA sequence of ATCC Deposit No:Z, which encodes a biologically active fragment of a polypeptide; (e) a nucleotide sequence in SEQ ID NO:X or the cDNA sequence of ATCC Deposit No:Z, which encodes an antigenic fragment of a polypeptide; (f) a nucleotide sequence encoding a polypeptide comprising the complete amino acid sequence of SEQ ID NO:Y or the complete amino acid sequence encoded by the cDNA in ATCC Deposit No:Z; (g) a nucleotide sequence encoding a mature polypeptide of the amino acid sequence of SEQ ID NO:Y (i.e., a secreted polypeptide (e.g., as delineated in columns 14 and 15 of Table 1A)) or a mature polypeptide of the amino acid sequence encoded by the cDNA in ATCC Deposit No:Z; (h) a nucleotide sequence encoding a biologically active fragment of a polypeptide having the complete amino acid sequence of SEO ID NO:Y or the complete amino acid sequence encoded by the cDNA in ATCC Deposit No:Z; (i) a nucleotide sequence encoding an antigenic fragment of a polypeptide having the complete amino acid sequence of SEQ ID NO:Y or the complete amino acid sequence encoded by the cDNA in ATCC Deposit No:Z; and (j) a nucleotide sequence complementary to any of the nucleotide sequences in (a), (b), (c), (d), (e), (f), (g), (h), or (i) above.

The present invention is also directed to nucleic acid molecules which comprise, or alternatively consist of, a nucleotide sequence which is at least 80%, 85%, 90%, 95%, 96%, 97%, 98%, 99% or 100%, identical to, for example, any of the nucleotide sequences in (a), (b), (c), (d), (e), (f), (g), (h), (i), or (j) above, the nucleotide coding sequence in SEQ ID NO:X or the complementary strand thereto, the nucleotide coding sequence of the cDNA contained in ATCC Deposit No:Z or the complementary strand thereto, a nucleotide sequence encoding the polypeptide of SEQ ID NO:Y, a nucleotide sequence encoding a polypeptide sequence encoded by the nucleotide sequence in SEQ ID NO:X, a polypeptide sequence encoded by the complement of the polynucleotide sequence in SEQ ID NO:X, a nucleotide sequence encoding the polypeptide encoded by the cDNA contained in ATCC Deposit No:Z, the nucleotide coding sequence in SEQ ID NO:X as defined in columns 8 and 9 of Table 2 or the complementary strand thereto, a nucleotide sequence encoding the polypeptide encoded by the nucleotide sequence in SEQ ID NO:X as defined in columns 8 and 9 of Table 2 or the complementary strand thereto, the nucleotide coding sequence in SEQ ID NO:B as defined in column 6 of Table 1C or the complementary strand thereto, a nucleotide sequence encoding the polypeptide encoded by the nucleotide sequence in SEQ ID NO:B as defined in column 6 of Table 1C or the complementary strand thereto, the nucleotide sequence in SEQ ID NO:X encoding the polypeptide sequence as

defined in Table 1B or the complementary strand thereto, nucleotide sequences encoding the polypeptide as defined in Table 1B or the complementary strand thereto, and/or polynucleotide fragments of any of these nucleic acid molecules (e.g., those fragments described herein). Polynucleotides which hybridize to the complement of these nucleic acid molecules under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention, as are polypeptides encoded by these polynucleotides and nucleic acids.

In a preferred embodiment, the invention encompasses nucleic acid molecules which comprise, or alternatively, consist of a polynucleotide which hybridizes under stringent hybridization conditions, or alternatively, under lower stringency conditions, to a polynucleotide in (a), (b), (c), (d), (e), (f), (g), (h), or (i), above, as are polypeptides encoded by these polynucleotides. In another preferred embodiment, polynucleotides which hybridize to the complement of these nucleic acid molecules under stringent hybridization conditions, or alternatively, under lower stringency conditions, are also encompassed by the invention, as are polypeptides encoded by these polynucleotides.

In another embodiment, the invention provides a purified protein comprising, or alternatively consisting of, a polypeptide having an amino acid sequence selected from the group consisting of: (a) the complete amino acid sequence of SEQ ID NO:Y or the complete amino acid sequence encoded by the cDNA in ATCC Deposit No:Z; (b) the amino acid sequence of a mature (secreted) form of a polypeptide having the amino acid sequence of SEQ ID NO:Y (e.g., as delineated in columns 14 and 15 of Table 1A) or a mature form of the amino acid sequence encoded by the cDNA in ATCC Deposit No:Z mature; (c) the amino acid sequence of a biologically active fragment of a polypeptide having the complete amino acid sequence of SEQ ID NO:Y or the complete amino acid sequence encoded by the cDNA in ATCC Deposit No:Z; and (d) the amino acid sequence of an antigenic fragment of a polypeptide having the complete amino acid sequence of SEQ ID NO:Y or the complete amino acid sequence encoded by the cDNA in ATCC Deposit No:Z.

The present invention is also directed to proteins which comprise, or alternatively consist of, an amino acid sequence which is at least 80%, 85%, 90%, 95%, 96%, 97%, 98%, 99% or 100%, identical to, for example, any of the amino acid sequences in (a), (b), (c), or (d), above, the amino acid sequence shown in SEQ ID NO:Y, the amino acid sequence encoded by the cDNA contained in ATCC Deposit No:Z, the amino acid sequence of the polypeptide encoded by the nucleotide sequence in SEQ ID NO:X as defined in columns 8 and 9 of Table 2, the amino acid sequence of the polypeptide encoded by the nucleotide sequence in SEQ ID NO:B as defined in column 6 of Table 1C, the amino acid sequence as defined in Table 1B, an amino acid sequence encoded by the nucleotide sequence in SEQ ID NO:X, and an amino acid sequence encoded by the complement of the polypucleotide sequence in SEQ ID NO:X. Fragments of these polypeptides

are also provided (e.g., those fragments described herein). Further proteins encoded by polynucleotides which hybridize to the complement of the nucleic acid molecules encoding these amino acid sequences under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention, as are the polynucleotides encoding these proteins.

By a nucleic acid having a nucleotide sequence at least, for example, 95% "identical" to a reference nucleotide sequence of the present invention, it is intended that the nucleotide sequence of the nucleic acid is identical to the reference sequence except that the nucleotide sequence may include up to five point mutations per each 100 nucleotides of the reference nucleotide sequence encoding the polypeptide. In other words, to obtain a nucleic acid having a nucleotide sequence at least 95% identical to a reference nucleotide sequence, up to 5% of the nucleotides in the reference sequence may be deleted or substituted with another nucleotide, or a number of nucleotides up to 5% of the total nucleotides in the reference sequence may be inserted into the reference sequence. The query sequence may be an entire sequence referred to in Table 1B or 2 as the ORF (open reading frame), or any fragment specified as described herein.

As a practical matter, whether any particular nucleic acid molecule or polypeptide is at least 80%, 85%, 90%, 95%, 96%, 97%, 98% or 99% identical to a nucleotide sequence of the present invention can be determined conventionally using known computer programs. A preferred method for determining the best overall match between a query sequence (a sequence of the present invention) and a subject sequence, also referred to as a global sequence alignment, can be determined using the FASTDB computer program based on the algorithm of Brutlag et al. (Comp. App. Biosci. 6:237-245 (1990)). In a sequence alignment the query and subject sequences are both DNA sequences. An RNA sequence can be compared by converting U's to T's. The result of said global sequence alignment is expressed as percent identity. Preferred parameters used in a FASTDB alignment of DNA sequences to calculate percent identity are: Matrix=Unitary, k-tuple=4, Mismatch Penalty=1, Joining Penalty=30, Randomization Group Length=0, Cutoff Score=1, Gap Penalty=5, Gap Size Penalty 0.05, Window Size=500 or the length of the subject nucleotide sequence, whichever is shorter.

If the subject sequence is shorter than the query sequence because of 5' or 3' deletions, not because of internal deletions, a manual correction must be made to the results. This is because the FASTDB program does not account for 5' and 3' truncations of the subject sequence when calculating percent identity. For subject sequences truncated at the 5' or 3' ends, relative to the query sequence, the percent identity is corrected by calculating the number of bases of the query sequence that are 5' and 3' of the subject sequence, which are not matched/aligned, as a percent of the total bases of the query sequence. Whether a nucleotide is matched/aligned is determined by results of the FASTDB sequence alignment. This percentage is then subtracted from the percent identity, calculated by the above FASTDB program using the specified parameters, to arrive at a

final percent identity score. This corrected score is what is used for the purposes of the present invention. Only bases outside the 5' and 3' bases of the subject sequence, as displayed by the FASTDB alignment, which are not matched/aligned with the query sequence, are calculated for the purposes of manually adjusting the percent identity score.

For example, a 90 base subject sequence is aligned to a 100 base query sequence to determine percent identity. The deletions occur at the 5' end of the subject sequence and therefore, the FASTDB alignment does not show a matched/alignment of the first 10 bases at 5' end. The 10 unpaired bases represent 10% of the sequence (number of bases at the 5' and 3' ends not matched/total number of bases in the query sequence) so 10% is subtracted from the percent identity score calculated by the FASTDB program. If the remaining 90 bases were perfectly matched the final percent identity would be 90%. In another example, a 90 base subject sequence is compared with a 100 base query sequence. This time the deletions are internal deletions so that there are no bases on the 5' or 3' of the subject sequence which are not matched/aligned with the query. In this case the percent identity calculated by FASTDB is not manually corrected. Once again, only bases 5' and 3' of the subject sequence which are not matched/aligned with the query sequence are manually corrected for. No other manual corrections are to be made for the purposes of the present invention.

By a polypeptide having an amino acid sequence at least, for example, 95% "identical" to a query amino acid sequence of the present invention, it is intended that the amino acid sequence of the subject polypeptide is identical to the query sequence except that the subject polypeptide sequence may include up to five amino acid alterations per each 100 amino acids of the query amino acid sequence. In other words, to obtain a polypeptide having an amino acid sequence at least 95% identical to a query amino acid sequence, up to 5% of the amino acid residues in the subject sequence may be inserted, deleted, (indels) or substituted with another amino acid. These alterations of the reference sequence may occur at the amino or carboxy terminal positions of the reference amino acid sequence or anywhere between those terminal positions, interspersed either individually among residues in the reference sequence or in one or more contiguous groups within the reference sequence.

As a practical matter, whether any particular polypeptide is at least 80%, 85%, 90%, 95%, 96%, 97%, 98% or 99% identical to, for instance, the amino acid sequence of a polypeptide referred to in Table 1A (e.g., the amino acid sequence delineated in columns 14 and 15) or a fragment thereof, Table 1B.1 (e.g., the amino acid sequence identified in column 6) or a fragment thereof, Table 2 (e.g., the amino acid sequence of the polypeptide encoded by the polynucleotide sequence defined in columns 8 and 9 of Table 2) or a fragment thereof, the amino acid sequence of the polypeptide encoded by the polynucleotide sequence in SEQ ID NO:B as defined in column 6 of Table 1C or a fragment thereof, the amino acid sequence of the polypeptide encoded by the nucleotide sequence in SEQ ID NO:X or a fragment thereof, or the amino acid sequence of the

polypeptide encoded by cDNA contained in ATCC Deposit No:Z, or a fragment thereof, the amino acid sequence of a mature (secreted) polypeptide encoded by cDNA contained in ATCC Deposit No:Z, or a fragment thereof, can be determined conventionally using known computer programs. A preferred method for determining the best overall match between a query sequence (a sequence of the present invention) and a subject sequence, also referred to as a global sequence alignment, can be determined using the FASTDB computer program based on the algorithm of Brutlag et al. (Comp. App. Biosci.6:237-245 (1990)). In a sequence alignment the query and subject sequences are either both nucleotide sequences or both amino acid sequences. The result of said global sequence alignment is expressed as percent identity. Preferred parameters used in a FASTDB amino acid alignment are: Matrix=PAM 0, k-tuple=2, Mismatch Penalty=1, Joining Penalty=20, Randomization Group Length=0, Cutoff Score=1, Window Size=sequence length, Gap Penalty=5, Gap Size Penalty=0.05, Window Size=500 or the length of the subject amino acid sequence, whichever is shorter.

If the subject sequence is shorter than the query sequence due to N- or C-terminal deletions, not because of internal deletions, a manual correction must be made to the results. This is because the FASTDB program does not account for N- and C-terminal truncations of the subject sequence when calculating global percent identity. For subject sequences truncated at the N- and C-termini, relative to the query sequence, the percent identity is corrected by calculating the number of residues of the query sequence that are N- and C-terminal of the subject sequence, which are not matched/aligned with a corresponding subject residue, as a percent of the total bases of the query sequence. Whether a residue is matched/aligned is determined by results of the FASTDB sequence alignment. This percentage is then subtracted from the percent identity, calculated by the above FASTDB program using the specified parameters, to arrive at a final percent identity score. This final percent identity score is what is used for the purposes of the present invention. Only residues to the N- and C-termini of the subject sequence, which are not matched/aligned with the query sequence, are considered for the purposes of manually adjusting the percent identity score. That is, only query residue positions outside the farthest N- and C-terminal residues of the subject sequence.

For example, a 90 amino acid residue subject sequence is aligned with a 100 residue query sequence to determine percent identity. The deletion occurs at the N-terminus of the subject sequence and therefore, the FASTDB alignment does not show a matching/alignment of the first 10 residues at the N-terminus. The 10 unpaired residues represent 10% of the sequence (number of residues at the N- and C- termini not matched/total number of residues in the query sequence) so 10% is subtracted from the percent identity score calculated by the FASTDB program. If the remaining 90 residues were perfectly matched the final percent identity would be 90%. In another example, a 90 residue subject sequence is compared with a 100 residue query sequence. This time the deletions are internal deletions so there are no residues at the N- or C-termini of the subject

sequence which are not matched/aligned with the query. In this case the percent identity calculated by FASTDB is not manually corrected. Once again, only residue positions outside the N- and C-terminal ends of the subject sequence, as displayed in the FASTDB alignment, which are not matched/aligned with the query sequence are manually corrected for. No other manual corrections are to made for the purposes of the present invention.

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The polynucleotide variants of the invention may contain alterations in the coding regions, non-coding regions, or both. Especially preferred are polynucleotide variants containing alterations which produce silent substitutions, additions, or deletions, but do not alter the properties or activities of the encoded polypeptide. Nucleotide variants produced by silent substitutions due to the degeneracy of the genetic code are preferred. Moreover, polypeptide variants in which less than 50, less than 40, less than 30, less than 20, less than 10, or 5-50, 5-25, 5-10, 1-5, or 1-2 amino acids are substituted, deleted, or added in any combination are also preferred. Polynucleotide variants can be produced for a variety of reasons, e.g., to optimize codon expression for a particular host (change codons in the human mRNA to those preferred by a bacterial host such as E. coli).

Naturally occurring variants are called "allelic variants," and refer to one of several alternate forms of a gene occupying a given locus on a chromosome of an organism. (Genes II, Lewin, B., ed., John Wiley & Sons, New York (1985)). These allelic variants can vary at either the polynucleotide and/or polypeptide level and are included in the present invention. Alternatively, non-naturally occurring variants may be produced by mutagenesis techniques or by direct synthesis.

Using known methods of protein engineering and recombinant DNA technology, variants may be generated to improve or alter the characteristics of the polypeptides of the present invention. For instance, one or more amino acids can be deleted from the N-terminus or C-terminus of the polypeptide of the present invention without substantial loss of biological function. As an example, Ron et al. (J. Biol. Chem. 268: 2984-2988 (1993)) reported variant KGF proteins having heparin binding activity even after deleting 3, 8, or 27 amino-terminal amino acid residues. Similarly, Interferon gamma exhibited up to ten times higher activity after deleting 8-10 amino acid residues from the carboxy terminus of this protein. (Dobeli et al., J. Biotechnology 7:199-216 (1988).)

Moreover, ample evidence demonstrates that variants often retain a biological activity similar to that of the naturally occurring protein. For example, Gayle and coworkers (J. Biol. Chem. 268:22105-22111 (1993)) conducted extensive mutational analysis of human cytokine IL-1a. They used random mutagenesis to generate over 3,500 individual IL-1a mutants that averaged 2.5 amino acid changes per variant over the entire length of the molecule. Multiple mutations were examined at every possible amino acid position. The investigators found that "[m]ost of the molecule could be altered with little effect on either [binding or biological activity]." In fact, only

23 unique amino acid sequences, out of more than 3,500 nucleotide sequences examined, produced a protein that significantly differed in activity from wild-type.

Furthermore, even if deleting one or more amino acids from the N-terminus or C-terminus of a polypeptide results in modification or loss of one or more biological functions, other biological activities may still be retained. For example, the ability of a deletion variant to induce and/or to bind antibodies which recognize the secreted form will likely be retained when less than the majority of the residues of the secreted form are removed from the N-terminus or C-terminus. Whether a particular polypeptide lacking N- or C-terminal residues of a protein retains such immunogenic activities can readily be determined by routine methods described herein and otherwise known in the art.

Thus, the invention further includes polypeptide variants which show a biological or functional activity of the polypeptides of the invention (such as, for example, activity useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders). Such variants include deletions, insertions, inversions, repeats, and substitutions selected according to general rules known in the art so as have little effect on activity.

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The present application is directed to nucleic acid molecules at least 80%, 85%, 90%, 95%, 96%, 97%, 98%, 99% or 100% identical to the nucleic acid sequences disclosed herein, (e.g., encoding a polypeptide having the amino acid sequence of an N and/or C terminal deletion), irrespective of whether they encode a polypeptide having functional activity. This is because even where a particular nucleic acid molecule does not encode a polypeptide having functional activity, one of skill in the art would still know how to use the nucleic acid molecule, for instance, as a hybridization probe or a polymerase chain reaction (PCR) primer. Uses of the nucleic acid molecules of the present invention that do not encode a polypeptide having functional activity include, inter alia, (1) isolating a gene or allelic or splice variants thereof in a cDNA library; (2) in situ hybridization (e.g., "FISH") to metaphase chromosomal spreads to provide precise chromosomal location of the gene, as described in Verma et al., Human Chromosomes: A Manual of Basic Techniques, Pergamon Press, New York (1988); (3) Northern Blot analysis for detecting mRNA expression in specific tissues (e.g., normal or diseased tissues); and (4) in situ hybridization (e.g., histochemistry) for detecting mRNA expression in specific tissues (e.g., normal or diseased tissues).

Preferred, however, are nucleic acid molecules having sequences at least 80%, 85%, 90%, 95%, 96%, 97%, 98%, 99% or 100% identical to the nucleic acid sequences disclosed herein, which do, in fact, encode a polypeptide having functional activity. By a polypeptide having "functional activity" is meant, a polypeptide capable of displaying one or more known functional activities associated with a full-length (complete) protein and/or a mature (secreted) protein of the invention. Such functional activities include, but are not limited to, biological activity (such as,

for example, activity useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders), antigenicity (ability to bind, or compete with a polypeptide of the invention for binding, to an anti-polypeptide of the invention antibody), immunogenicity (ability to generate antibody which binds to a specific polypeptide of the invention), ability to form multimers with polypeptides of the invention, and ability to bind to a receptor or ligand for a polypeptide of the invention.

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The functional activity of the polypeptides, and fragments, variants and derivatives of the invention, can be assayed by various methods.

For example, in one embodiment where one is assaying for the ability to bind or compete with a full-length polypeptide of the present invention for binding to an anti-polypetide antibody, various immunoassays known in the art can be used, including but not limited to, competitive and non-competitive assay systems using techniques such as radioimmunoassays, ELISA (enzyme linked immunosorbent assay), "sandwich" immunoassays, immunoradiometric assays, gel diffusion precipitation reactions, immunodiffusion assays, in situ immunoassays (using colloidal gold, enzyme or radioisotope labels, for example), western blots, precipitation reactions, agglutination assays (e.g., gel agglutination assays, hemagglutination assays), complement fixation assays, immunofluorescence assays, protein A assays, and immunoelectrophoresis assays, etc. In one embodiment, antibody binding is detected by detecting a label on the primary antibody. In another embodiment, the primary antibody is detected by detecting binding of a secondary antibody or reagent to the primary antibody. In a further embodiment, the secondary antibody is labeled. Many means are known in the art for detecting binding in an immunoassay and are within the scope of the present invention.

In another embodiment, where a ligand is identified, or the ability of a polypeptide fragment, variant or derivative of the invention to multimerize is being evaluated, binding can be assayed, e.g., by means well-known in the art, such as, for example, reducing and non-reducing gel chromatography, protein affinity chromatography, and affinity blotting. See generally, Phizicky et al., Microbiol. Rev. 59:94-123 (1995). In another embodiment, the ability of physiological correlates of a polypeptide of the present invention to bind to a substrate(s) of the polypeptide of the invention can be routinely assayed using techniques known in the art.

In addition, assays described herein (see Examples) and otherwise known in the art may routinely be applied to measure the ability of polypeptides of the present invention and fragments, variants and derivatives thereof to elicit polypeptide related biological activity (either *in vitro* or *in vivo*). Other methods will be known to the skilled artisan and are within the scope of the invention.

Of course, due to the degeneracy of the genetic code, one of ordinary skill in the art will immediately recognize that a large number of the nucleic acid molecules having a sequence at least 80%, 85%, 90%, 95%, 96%, 97%, 98%, 99%, or 100% identical to, for example, the nucleic

acid sequence of the cDNA contained in ATCC Deposit No:Z, the nucleic acid sequence referred to in Table 1B (SEQ ID NO:X), the nucleic acid sequence disclosed in Table 1A (e.g., the nucleic acid sequence delineated in columns 7 and 8), the nucleic acid sequence disclosed in Table 2 (e.g., the nucleic acid sequence delineated in columns 8 and 9) or fragments thereof, will encode polypeptides "having functional activity." In fact, since degenerate variants of any of these nucleotide sequences all encode the same polypeptide, in many instances, this will be clear to the skilled artisan even without performing the above described comparison assay. It will be further recognized in the art that, for such nucleic acid molecules that are not degenerate variants, a reasonable number will also encode a polypeptide having functional activity. This is because the skilled artisan is fully aware of amino acid substitutions that are either less likely or not likely to significantly effect protein function (e.g., replacing one aliphatic amino acid with a second aliphatic amino acid), as further described below.

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For example, guidance concerning how to make phenotypically silent amino acid substitutions is provided in Bowie et al., "Deciphering the Message in Protein Sequences: Tolerance to Amino Acid Substitutions," Science 247:1306-1310 (1990), wherein the authors indicate that there are two main strategies for studying the tolerance of an amino acid sequence to change.

The first strategy exploits the tolerance of amino acid substitutions by natural selection during the process of evolution. By comparing amino acid sequences in different species, conserved amino acids can be identified. These conserved amino acids are likely important for protein function. In contrast, the amino acid positions where substitutions have been tolerated by natural selection indicates that these positions are not critical for protein function. Thus, positions tolerating amino acid substitution could be modified while still maintaining biological activity of the protein.

The second strategy uses genetic engineering to introduce amino acid changes at specific positions of a cloned gene to identify regions critical for protein function. For example, site directed mutagenesis or alanine-scanning mutagenesis (introduction of single alanine mutations at every residue in the molecule) can be used. See Cunningham and Wells, Science 244:1081-1085 (1989). The resulting mutant molecules can then be tested for biological activity.

As the authors state, these two strategies have revealed that proteins are surprisingly tolerant of amino acid substitutions. The authors further indicate which amino acid changes are likely to be permissive at certain amino acid positions in the protein. For example, most buried (within the tertiary structure of the protein) amino acid residues require nonpolar side chains, whereas few features of surface side chains are generally conserved. Moreover, tolerated conservative amino acid substitutions involve replacement of the aliphatic or hydrophobic amino acids Ala, Val, Leu and Ile; replacement of the hydroxyl residues Ser and Thr; replacement of the acidic residues Asp and Glu; replacement of the amide residues Asp and Glu, replacement of the

basic residues Lys, Arg, and His; replacement of the aromatic residues Phe, Tyr, and Trp, and replacement of the small-sized amino acids Ala, Ser, Thr, Met, and Gly.

Besides conservative amino acid substitution, variants of the present invention include (i) substitutions with one or more of the non-conserved amino acid residues, where the substituted amino acid residues may or may not be one encoded by the genetic code, or (ii) substitutions with one or more of the amino acid residues having a substituent group, or (iii) fusion of the mature polypeptide with another compound, such as a compound to increase the stability and/or solubility of the polypeptide (for example, polyethylene glycol), (iv) fusion of the polypeptide with additional amino acids, such as, for example, an IgG Fc fusion region peptide, serum albumin (preferably human serum albumin) or a fragment thereof, or leader or secretory sequence, or a sequence facilitating purification, or (v) fusion of the polypeptide with another compound, such as albumin (including but not limited to recombinant albumin (see, e.g., U.S. Patent No. 5,876,969, issued March 2, 1999, EP Patent 0 413 622, and U.S. Patent No. 5,766,883, issued June 16, 1998, herein incorporated by reference in their entirety)). Such variant polypeptides are deemed to be within the scope of those skilled in the art from the teachings herein.

For example, polypeptide variants containing amino acid substitutions of charged amino acids with other charged or neutral amino acids may produce proteins with improved characteristics, such as less aggregation. Aggregation of pharmaceutical formulations both reduces activity and increases clearance due to the aggregate's immunogenic activity. See Pinckard et al., Clin. Exp. Immunol. 2:331-340 (1967); Robbins et al., Diabetes 36: 838-845 (1987); Cleland et al., Crit. Rev. Therapeutic Drug Carrier Systems 10:307-377 (1993).

A further embodiment of the invention relates to polypeptides which comprise the amino acid sequence of a polypeptide having an amino acid sequence which contains at least one amino acid substitution, but not more than 50 amino acid substitutions, even more preferably, not more than 40 amino acid substitutions, still more preferably, not more than 30 amino acid substitutions, and still even more preferably, not more than 20 amino acid substitutions from a polypeptide sequence disclosed herein. Of course it is highly preferable for a polypeptide to have an amino acid sequence which, for example, comprises the amino acid sequence of a polypeptide of SEQ ID NO:Y, the amino acid sequence of the mature (e.g., secreted) polypeptide of SEQ ID NO:Y, an amino acid sequence encoded by SEQ ID NO:X, an amino acid sequence encoded by the portion of SEQ ID NO:X as defined in columnns 8 and 9 of Table 2, an amino acid sequence encoded by the complement of SEQ ID NO:X, an amino acid sequence encoded by cDNA contained in ATCC Deposit No:Z, and/or the amino acid sequence of a mature (secreted) polypeptide encoded by cDNA contained in ATCC Deposit No:Z, or a fragment thereof, which contains, in order of everincreasing preference, at least one, but not more than 10, 9, 8, 7, 6, 5, 4, 3, 2 or 1 amino acid substitutions.

In specific embodiments, the polypeptides of the invention comprise, or alternatively, consist of, fragments or variants of a reference amino acid sequence selected from: (a) the amino acid sequence of SEQ ID NO:Y or fragments thereof (e.g., the mature formand/or other fragments described herein); (b) the amino acid sequence encoded by SEQ ID NO:X or fragments thereof; (c) the amino acid sequence encoded by the complement of SEQ ID NO:X or fragments thereof; (d) the amino acid sequence encoded by the portion of SEQ ID NO:X as defined in columns 8 and 9 of Table 2 or fragments thereof; and (e) the amino acid sequence encoded by cDNA contained in ATCC Deposit No:Z or fragments thereof; wherein the fragments or variants have 1-5, 5-10, 5-25, 5-50, 10-50 or 50-150, amino acid residue additions, substitutions, and/or deletions when compared to the reference amino acid sequence. In preferred embodiments, the amino acid substitutions are conservative. Polynucleotides encoding these polypeptides are also encompassed by the invention.

## Polynucleotide and Polypeptide Fragments

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The present invention is also directed to polynucleotide fragments of the polynucleotides (nucleic acids) of the invention. In the present invention, a "polynucleotide fragment" refers to a polynucleotide having a nucleic acid sequence which, for example: is a portion of the cDNA contained in ATCC Deposit No:Z or the complementary strand thereto; is a portion of the polynucleotide sequence encoding the polypeptide encoded by the cDNA contained in ATCC Deposit No:Z or the complementary strand thereto; is a portion of the polynucleotide sequence encoding the mature (secreted) polypeptide encoded by the cDNA contained in ATCC Deposit No:Z or the complementary strand thereto; is a portion of a polynucleotide sequence encoding the mature amino acid sequence as defined in columns 14 and 15 of Table 1A or the complementary strand thereto; is a portion of a polynucleotide sequence encoding the amino acid sequence encoded by the region of SEQ ID NO:X as defined in columns 8 and 9 of Table 2 or the complementary strand thereto; is a portion of the polynucleotide sequence of SEQ ID NO:X as defined in columns 8 and 9 of Table 2 or the complementary strand thereto; is a portion of the polynucleotide sequence in SEQ ID NO:X or the complementary strand thereto; is a polynucleotide sequence encoding a portion of the polypeptide of SEQ ID NO:Y; is a polynucleotide sequence encoding a portion of a polypeptide encoded by SEQ ID NO:X; is a polynucleotide sequence encoding a portion of a polypeptide encoded by the complement of the polynucleotide sequence in SEQ ID NO:X; is a portion of a polynucleotide sequence encoding the amino acid sequence encoded by the region of SEQ ID NO:B as defined in column 6 of Table 1C or the complementary strand thereto; or is a portion of the polynucleotide sequence of SEQ ID NO:B as defined in column 6 of Table 1C or the complementary strand thereto.

The polynucleotide fragments of the invention are preferably at least about 15 nt, and more preferably at least about 20 nt, still more preferably at least about 30 nt, and even more

preferably, at least about 40 nt, at least about 50 nt, at least about 75 nt, or at least about 150 nt in length. A fragment "at least 20 nt in length," for example, is intended to include 20 or more contiguous bases from the cDNA sequence contained in ATCC Deposit No:Z, or the nucleotide sequence shown in SEQ ID NO:X or the complementary stand thereto. In this context "about" includes the particularly recited value or a value larger or smaller by several (5, 4, 3, 2, or 1) nucleotides, at either terminus or at both termini. These nucleotide fragments have uses that include, but are not limited to, as diagnostic probes and primers as discussed herein. Of course, larger fragments (e.g., at least 160, 170, 180, 190, 200, 250, 500, 600, 1000, or 2000 nucleotides in length) are also encompassed by the invention.

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Moreover, representative examples of polynucleotide fragments of the invention comprise, or alternatively consist of, a sequence from about nucleotide number 1-50, 51-100, 101-150, 151-200. 201-250. 251-300. 301-350. 351-400. 401-450. 451-500. 501-550. 551-600. 601-650. 651-700, 701-750, 751-800, 801-850, 851-900, 901-950, 951-1000, 1001-1050, 1051-1100, 1101-1150, 1151-1200, 1201-1250, 1251-1300, 1301-1350, 1351-1400, 1401-1450, 1451-1500, 1501-1550, 1551-1600, 1601-1650, 1651-1700, 1701-1750, 1751-1800, 1801-1850, 1851-1900, 1901-1950, 1951-2000, 2001-2050, 2051-2100, 2101-2150, 2151-2200, 2201-2250, 2251-2300, 2301-2350, 2351-2400, 2401-2450, 2451-2500, 2501-2550, 2551-2600, 2601-2650, 2651-2700, 2701-2750, 2751-2800, 2801-2850, 2851-2900, 2901-2950, 2951-3000, 3001-3050, 3051-3100, 3101-3150, 3151-3200, 3201-3250, 3251-3300, 3301-3350, 3351-3400, 3401-3450, 3451-3500, 3501-3550, 3551-3600, 3601-3650, 3651-3700, 3701-3750, 3751-3800, 3801-3850, 3851-3900, 3901-3950, 3951-4000, 4001-4050, 4051-4100, 4101-4150, 4151-4200, 4201-4250, 4251-4300, 4301-4350, 4351-4400, 4401-4450, 4451-4500, 4501-4550, 4551-4600, 4601-4650, 4651-4700, 4701-4750, 4751-4800, 4801-4850, 4851-4900, 4901-4950, 4951-5000, 5001-5050, 5051-5100, 5101-5150, 5151-5200, 5201-5250, 5251-5300, 5301-5350, 5351-5400, 5401-5450, 5451-5500, 5501-5550, 5551-5600, 5601-5650, 5651-5700, 5701-5750, 5751-5800, 5801-5850, 5851-5900, 5901-5950, 5951-6000, 6001-6050, 6051-6100, 6101-6150, 6151-6200, 6201-6250, 6251-6300, 6301-6350, 6351-6400, 6401-6450, 6451-6500, 6501-6550, 6551-6600, 6601-6650, 6651-6700, 6701-6750, 6751-6800, 6801-6850, 6851-6900, 6901-6950, 6951-7000, 7001-7050, 7051-7100, 7101-7150, 7151-7200, 7201-7250, 7251-7300 or 7301 to the end of SEQ ID NO:X, or the complementary strand thereto. In this context "about" includes the particularly recited range or a range larger or smaller by several (5, 4, 3, 2, or 1) nucleotides, at either terminus or at both termini. Preferably, these fragments encode a polypeptide which has a functional activity (e.g., biological activity; such as, for example, activity useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders). More preferably, these polynucleotides can be used as probes or primers as discussed herein. Polynucleotides which hybridize to one or more of these polynucleotides under stringent

hybridization conditions or alternatively, under lower stringency conditions are also encompassed by the invention, as are polypeptides encoded by these polynucleotides.

Further representative examples of polynucleotide fragments of the invention comprise, or alternatively consist of, a sequence from about nucleotide number 1-50, 51-100, 101-150, 151-200, 201-250, 251-300, 301-350, 351-400, 401-450, 451-500, 501-550, 551-600, 601-650, 651-5 700, 701-750, 751-800, 801-850, 851-900, 901-950, 951-1000, 1001-1050, 1051-1100, 1101-1150, 1151-1200, 1201-1250, 1251-1300, 1301-1350, 1351-1400, 1401-1450, 1451-1500, 1501-1550, 1551-1600, 1601-1650, 1651-1700, 1701-1750, 1751-1800, 1801-1850, 1851-1900, 1901-1950, 1951-2000, 2001-2050, 2051-2100, 2101-2150, 2151-2200, 2201-2250, 2251-2300, 2301-2350, 2351-2400, 2401-2450, 2451-2500, 2501-2550, 2551-2600, 2601-2650, 2651-2700, 2701-10 2750, 2751-2800, 2801-2850, 2851-2900, 2901-2950, 2951-3000, 3001-3050, 3051-3100, 3101-3150, 3151-3200, 3201-3250, 3251-3300, 3301-3350, 3351-3400, 3401-3450, 3451-3500, 3501-3550, 3551-3600, 3601-3650, 3651-3700, 3701-3750, 3751-3800, 3801-3850, 3851-3900, 3901-3950, 3951-4000, 4001-4050, 4051-4100, 4101-4150, 4151-4200, 4201-4250, 4251-4300, 4301-4350, 4351-4400, 4401-4450, 4451-4500, 4501-4550, 4551-4600, 4601-4650, 4651-4700, 4701-15 4750, 4751-4800, 4801-4850, 4851-4900, 4901-4950, 4951-5000, 5001-5050, 5051-5100, 5101-5150, 5151-5200, 5201-5250, 5251-5300, 5301-5350, 5351-5400, 5401-5450, 5451-5500, 5501-5550, 5551-5600, 5601-5650, 5651-5700, 5701-5750, 5751-5800, 5801-5850, 5851-5900, 5901-5950, 5951-6000, 6001-6050, 6051-6100, 6101-6150, 6151-6200, 6201-6250, 6251-6300, 6301-6350, 6351-6400, 6401-6450, 6451-6500, 6501-6550, 6551-6600, 6601-6650, 6651-6700, 6701-20 6750, 6751-6800, 6801-6850, 6851-6900, 6901-6950, 6951-7000, 7001-7050, 7051-7100, 7101-7150, 7151-7200, 7201-7250, 7251-7300 or 7301 to the end of the cDNA sequence contained in ATCC Deposit No:Z, or the complementary strand thereto. In this context "about" includes the particularly recited range or a range larger or smaller by several (5, 4, 3, 2, or 1) nucleotides, at either terminus or at both termini. Preferably, these fragments encode a polypeptide which has a 25 functional activity (e.g., biological activity). More preferably, these polynucleotides can be used as probes or primers as discussed herein. Polynucleotides which hybridize to one or more of these polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions are also encompassed by the invention, as are polypeptides encoded by these 30 polynucleotides.

Moreover, representative examples of polynucleotide fragments of the invention comprise, or alternatively consist of, a nucleic acid sequence comprising one, two, three, four, five, six, seven, eight, nine, ten, or more of the above described polynucleotide fragments of the invention in combination with a polynucleotide sequence delineated in Table 1C column 6. Additional, representative examples of polynucleotide fragments of the invention comprise, or alternatively consist of, a nucleic acid sequence comprising one, two, three, four, five, six, seven, eight, nine, ten, or more of the above described polynucleotide fragments of the invention in combination with

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a polynucleotide sequence that is the complementary strand of a sequence delineated in column 6 of Table 1C. In further embodiments, the above-described polynucleotide fragments of the invention comprise, or alternatively consist of, sequences delineated in Table 1C, column 6, and have a nucleic acid sequence which is different from that of the BAC fragment having the sequence disclosed in SEQ ID NO:B (see Table 1C, column 5). In additional embodiments, the above-described polynucleotide fragments of the invention comprise, or alternatively consist of, sequences delineated in Table 1C, column 6, and have a nucleic acid sequence which is different from that published for the BAC clone identified as BAC ID NO:A (see Table 1C, column 4). In additional embodiments, the above-described polynucleotides of the invention comprise, or alternatively consist of, sequences delineated Table 1C, column 6, and have a nucleic acid sequence which is different from that contained in the BAC clone identified as BAC ID NO:A (see Table 1C, column 4). Polypeptides encoded by these polynucleotides, other polynucleotides that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides and polypeptides are also encompassed by the invention.

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In additional specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more fragments of the sequences delineated in column 6 of Table 1C, and the polynucleotide sequence of SEQ ID NO:X (e.g., as defined in Table 1C, column 2) or fragments or variants thereof. Polypeptides encoded by these polynucleotides, other polynucleotides that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention.

In additional specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more fragments of the sequences delineated in column 6 of Table 1C which correspond to the same ATCC Deposit No:Z (see Table 1C, column 1), and the polynucleotide sequence of SEQ ID NO:X (e.g., as defined in Table 1A, 1B, or 1C) or fragments or variants thereof. Polypeptides encoded by these polynucleotides, other polynucleotides that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention.

In further specific embodiments, polynucleotides of the invention comprise, or alternatively consist of, one, two, three, four, five, six, seven, eight, nine, ten, or more fragments of the sequences delineated in the same row of column 6 of Table 1C, and the polynucleotide sequence of SEQ ID NO:X (e.g., as defined in Table 1A, 1B, or 1C) or fragments or variants thereof. Polypeptides encoded by these polynucleotides, other polynucleotides that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention.

In additional specific embodiments, polynucleotides of the invention comprise, or alternatively consist of a polynucleotide sequence in which the 3' 10 polynucleotides of one of the sequences delineated in column 6 of Table 1C and the 5' 10 polynucleotides of the sequence of

SEQ ID NO:X are directly contiguous. Nucleic acids which hybridize to the complement of these 20 contiguous polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or nucleic acids that encode these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides are also encompassed by the invention.

In additional specific embodiments, polynucleotides of the invention comprise, or alternatively consist of a polynucleotide sequence in which the 3' 10 polynucleotides of one of the sequences delineated in column 6 of Table 1C and the 5' 10 polynucleotides of a fragment or variant of the sequence of SEQ ID NO:X (e.g., as described herein) are directly contiguous Nucleic acids which hybridize to the complement of these 20 contiguous polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or nucleic acids encoding these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides are also encompassed by the invention.

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In further specific embodiments, polynucleotides of the invention comprise, or alternatively consist of a polynucleotide sequence in which the 3' 10 polynucleotides of a fragment or variant of the sequence of SEQ ID NO:X and the 5' 10 polynucleotides of the sequence of one of the sequences delineated in column 6 of Table 1C are directly contiguous. Nucleic acids which hybridize to the complement of these 20 contiguous polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or nucleic acids encoding these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides are also encompassed by the invention.

In specific embodiments, polynucleotides of the invention comprise, or alternatively consist of a polynucleotide sequence in which the 3' 10 polynucleotides of one of the sequences delineated in column 6 of Table 1C and the 5' 10 polynucleotides of another sequence in column 6 are directly contiguous. In preferred embodiments, the 3' 10 polynucleotides of one of the sequences delineated in column 6 of Table 1C is directly contiguous with the 5' 10 polynucleotides of the next sequential exon delineated in Table 1C, column 6. Nucleic acids which hybridize to the complement of these 20 contiguous polynucleotides under stringent hybridization conditions or alternatively, under lower stringency conditions, are also encompassed

by the invention. Polypeptides encoded by these polynucleotides and/or nucleic acids, other polynucleotides and/or nucleic acids encoding these polypeptides, and antibodies that bind these polypeptides are also encompassed by the invention. Additionally, fragments and variants of the above-described polynucleotides, nucleic acids, and polypeptides are also encompassed by the invention.

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In the present invention, a "polypeptide fragment" refers to an amino acid sequence which is a portion of the amino acid sequence contained in SEQ ID NO:Y, is a portion of the mature form of SEQ ID NO:Y as defined in columns 14 and 15 of Table 1A, a portion of an amino acid sequence encoded by the portion of SEQ ID NO:X as defined in columnns 8 and 9 of Table 2, is a portion of an amino acid sequence encoded by the polynucleotide sequence of SEQ ID NO:X, is a portion of an amino acid sequence encoded by the complement of the polynucleotide sequence in SEO ID NO:X, is a portion of the amino acid sequence of a mature (secreted) polypeptide encoded by the cDNA contained in ATCC Deposit No:Z, and/or is a portion of an amino acid sequence encoded by the cDNA contained in ATCC Deposit No:Z. Protein (polypeptide) fragments may be "free-standing," or comprised within a larger polypeptide of which the fragment forms a part or region, most preferably as a single continuous region. Representative examples of polypeptide fragments of the invention, include, for example, fragments comprising, or alternatively consisting of, from about amino acid number 1-20, 21-40, 41-60, 61-80, 81-100, 101-120, 121-140, 141-160, 161-180, 181-200, 201-220, 221-240, 241-260, 261-280, 281-300, 301-320, 321-340, 341-360, 361-380, 381-400, 401-420, 421-440, 441-460, 461-480, 481-500, 501-520, 521-540, 541-560, 561-580, 581-600, 601-620, 621-640, 641-660, 661-680, 681-700, 701-720, 721-740, 741-760, 761-780, 781-800, 801-820, 821-840, 841-860, 861-880, 881-900, 901-920, 921-940, 941-960, 961-980, 981-1000, 1001-1020, 1021-1040, 1041-1060, 1061-1080, 1081-1100, 1101-1120, 1121-1140, 1141-1160, 1161-1180, 1181-1200, 1201-1220, 1221-1240, 1241-1260, 1261-1280, 1281-1300, 1301-1320, 1321-1340, 1341-1360, 1361-1380, 1381-1400, 1401-1420, 1421-1440, or 1441 to the end of the coding region of cDNA and SEQ ID NO: Y. In a preferred embodiment, polypeptide fragments of the invention include, for example, fragments comprising, or alternatively consisting of, from about amino acid number 1-20, 21-40, 41-60, 61-80, 81-100, 101-120, 121-140, 141-160, 161-180, 181-200, 201-220, 221-240, 241-260, 261-280, 281-300, 301-320, 321-340, 341-360, 361-380, 381-400, 401-420, 421-440, 441-460, 461-480, 481-500, 501-520, 521-540, 541-560, 561-580, 581-600, 601-620, 621-640, 641-660, 661-680, 681-700, 701-720, 721-740, 741-760, 761-780, 781-800, 801-820, 821-840, 841-860, 861-880, 881-900, 901-920, 921-940, 941-960, 961-980, 981-1000, 1001-1020, 1021-1040, 1041-1060, 1061-1080, 1081-1100, 1101-1120, 1121-1140, 1141-1160, 1161-1180, 1181-1200, 1201-1220, 1221-1240, 1241-1260, 1261-1280, 1281-1300, 1301-1320, 1321-1340, 1341-1360, 1361-1380, 1381-1400, 1401-1420, 1421-1440, or 1441 to the end of the coding region of SEQ ID NO:Y. Moreover, polypeptide fragments of the invention may be at least about 10, 15, 20, 25, 30, 35, 40, 45, 50, 55,

60, 65, 70, 75, 80, 85, 90, 100, 110, 120, 130, 140, or 150 amino acids in length. In this context "about" includes the particularly recited ranges or values, or ranges or values larger or smaller by several (5, 4, 3, 2, or 1) amino acids, at either extreme or at both extremes. Polynucleotides encoding these polypeptide fragments are also encompassed by the invention.

Even if deletion of one or more amino acids from the N-terminus of a protein results in modification of loss of one or more biological functions of the protein, other functional activities (e.g., biological activities; such as, for example, activity useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders; ability to multimerize; ability to bind a ligand; antigenic ability useful for production of polypeptide specific antibodies) may still be retained. For example, the ability of shortened muteins to induce and/or bind to antibodies which recognize the complete or mature forms of the polypeptides generally will be retained when less than the majority of the residues of the complete or mature polypeptide are removed from the N-terminus. Whether a particular polypeptide lacking N-terminal residues of a complete polypeptide retains such immunologic activities can readily be determined by routine methods described herein and otherwise known in the art. It is not unlikely that a mutein with a large number of deleted N-terminal amino acid residues may retain some biological or immunogenic activities. In fact, peptides composed of as few as six amino acid residues may often evoke an immune response.

Accordingly, polypeptide fragments include the secreted protein as well as the mature form. Further preferred polypeptide fragments include the secreted protein or the mature form having a continuous series of deleted residues from the amino or the carboxy terminus, or both. For example, any number of amino acids, ranging from 1-60, can be deleted from the amino terminus of either the secreted polypeptide or the mature form. Similarly, any number of amino acids, ranging from 1-30, can be deleted from the carboxy terminus of the secreted protein or mature form. Furthermore, any combination of the above amino and carboxy terminus deletions are preferred. Similarly, polynucleotides encoding these polypeptide fragments are also preferred.

The present invention further provides polypeptides having one or more residues deleted from the amino terminus of the amino acid sequence of a polypeptide disclosed herein (e.g., a polypeptide of SEQ ID NO:Y, a polypeptide as defined in columns 14 and 15 of Table 1A, a polypeptide encoded by the polynucleotide sequence contained in SEQ ID NO:X or the complement thereof, a polypeptide encoded by the portion of SEQ ID NO:X as defined in columns 8 and 9 of Table 2, a polypeptide encoded by the portion of SEQ ID NO:B as defined in column 6 of Table 1C, a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z, and/or a mature polypeptide encoded by the cDNA contained in ATCC Deposit No:Z). In particular, N-terminal deletions may be described by the general formula m-q, where q is a whole integer representing the total number of amino acid residues in a polypeptide of the invention (e.g., the polypeptide disclosed in SEQ ID NO:Y, the mature (secreted) portion of SEQ ID NO:Y as defined

in columns 14 and 15 of Table 1A, or the polypeptide encoded by the portion of SEQ ID NO:X as defined in columns 8 and 9 of Table 2), and m is defined as any integer ranging from 2 to q-6. Polynucleotides encoding these polypeptides are also encompassed by the invention.

The present invention further provides polypeptides having one or more residues from the carboxy terminus of the amino acid sequence of a polypeptide disclosed herein (e.g., a polypeptide of SEQ ID NO:Y, the mature (secreted) portion of SEQ ID NO:Y as defined in columns 14 and 15 of Table 1A, a polypeptide encoded by the polynucleotide sequence contained in SEQ ID NO:X, a polypeptide encoded by the portion of SEQ ID NO:X as defined in columns 8 and 9 of Table 2, a polypeptide encoded by the portion of SEQ ID NO:B as defined in column 6 of Table 1C, a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z, and/or a mature polypeptide encoded by the cDNA contained in ATCC Deposit No:Z). In particular, C-terminal deletions may be described by the general formula 1-n, where n is any whole integer ranging from 6 to q-1, and where n corresponds to the position of amino acid residue in a polypeptide of the invention. Polynucleotides encoding these polypeptides are also encompassed by the invention.

In addition, any of the above described N- or C-terminal deletions can be combined to produce a N- and C-terminal deleted polypeptide. The invention also provides polypeptides having one or more amino acids deleted from both the amino and the carboxyl termini, which may be described generally as having residues m-n of a polypeptide encoded by SEQ ID NO:X (e.g., including, but not limited to, the preferred polypeptide disclosed as SEQ ID NO:Y, the mature (secreted) portion of SEQ ID NO:Y as defined in columns 14 and 15 of Table 1A, and the polypeptide encoded by the portion of SEQ ID NO:X as defined in columns 8 and 9 of Table 2), the cDNA contained in ATCC Deposit No:Z, and/or the complement thereof, where n and m are integers as described above. Polynucleotides encoding these polypeptides are also encompassed by the invention.

Also as mentioned above, even if deletion of one or more amino acids from the C-terminus of a protein results in modification of loss of one or more biological functions of the protein, other functional activities (e.g., biological activities such as, for example, activity useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders; ability to multimerize; ability to bind a ligand; antigenic ability useful for production of polypeptide specific antibodies) may still be retained. For example the ability of the shortened mutein to induce and/or bind to antibodies which recognize the complete or mature forms of the polypeptide generally will be retained when less than the majority of the residues of the complete or mature polypeptide are removed from the C-terminus. Whether a particular polypeptide lacking C-terminal residues of a complete polypeptide retains such immunologic activities can readily be determined by routine methods described herein and otherwise known in the art. It is not unlikely that a mutein with a large number of deleted

C-terminal amino acid residues may retain some biological or immunogenic activities. In fact, peptides composed of as few as six amino acid residues may often evoke an immune response.

The present application is also directed to proteins containing polypeptides at least 80%, 85%, 90%, 95%, 96%, 97%, 98% or 99% identical to a polypeptide sequence set forth herein. In preferred embodiments, the application is directed to proteins containing polypeptides at least 80%, 85%, 90%, 95%, 96%, 97%, 98% or 99% identical to polypeptides having the amino acid sequence of the specific N- and C-terminal deletions. Polynucleotides encoding these polypeptides are also encompassed by the invention.

Any polypeptide sequence encoded by, for example, the polynucleotide sequences set forth as SEQ ID NO:X or the complement thereof, (presented, for example, in Tables 1A and 2), the cDNA contained in ATCC Deposit No:Z, or the polynucleotide sequence as defined in column 6 of Table 1C, may be analyzed to determine certain preferred regions of the polypeptide. For example, the amino acid sequence of a polypeptide encoded by a polynucleotide sequence of SEQ ID NO:X (e.g., the polypeptide of SEQ ID NO:Y and the polypeptide encoded by the portion of SEQ ID NO:X as defined in columnns 8 and 9 of Table 2) or the cDNA contained in ATCC Deposit No:Z may be analyzed using the default parameters of the DNASTAR computer algorithm (DNASTAR, Inc., 1228 S. Park St., Madison, WI 53715 USA; http://www.dnastar.com/).

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Polypeptide regions that may be routinely obtained using the DNASTAR computer algorithm include, but are not limited to, Garnier-Robson alpha-regions, beta-regions, turn-regions, and coil-regions; Chou-Fasman alpha-regions, beta-regions, and turn-regions; Kyte-Doolittle hydrophilic regions and hydrophobic regions; Eisenberg alpha- and beta-amphipathic regions; Karplus-Schulz flexible regions; Emini surface-forming regions; and Jameson-Wolf regions of high antigenic index. Among highly preferred polynucleotides of the invention in this regard are those that encode polypeptides comprising regions that combine several structural features, such as several (e.g., 1, 2, 3 or 4) of the features set out above.

Additionally, Kyte-Doolittle hydrophilic regions and hydrophobic regions, Emini surface-forming regions, and Jameson-Wolf regions of high antigenic index (i.e., containing four or more contiguous amino acids having an antigenic index of greater than or equal to 1.5, as identified using the default parameters of the Jameson-Wolf program) can routinely be used to determine polypeptide regions that exhibit a high degree of potential for antigenicity. Regions of high antigenicity are determined from data by DNASTAR analysis by choosing values which represent regions of the polypeptide which are likely to be exposed on the surface of the polypeptide in an environment in which antigen recognition may occur in the process of initiation of an immune response.

Preferred polypeptide fragments of the invention are fragments comprising, or alternatively, consisting of, an amino acid sequence that displays a functional activity (e.g.

biological activity such as, for example, activity useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hematopoietic and hematologic diseases and disorders; ability to multimerize; ability to bind a ligand; antigenic ability useful for production of polypeptide specific antibodies) of the polypeptide sequence of which the amino acid sequence is a fragment. By a polypeptide displaying a "functional activity" is meant a polypeptide capable of one or more known functional activities associated with a full-length protein, such as, for example, biological activity, antigenicity, immunogenicity, and/or multimerization, as described herein.

Other preferred polypeptide fragments are biologically active fragments. Biologically active fragments are those exhibiting activity similar, but not necessarily identical, to an activity of the polypeptide of the present invention. The biological activity of the fragments may include an improved desired activity, or a decreased undesirable activity.

In preferred embodiments, polypeptides of the invention comprise, or alternatively consist of, one, two, three, four, five or more of the antigenic fragments of the polypeptide of SEQ ID NO:Y, or portions thereof. Polynucleotides encoding these polypeptides are also encompassed by the invention.

## Epitopes and Antibodies

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The present invention encompasses polypeptides comprising, or alternatively consisting of, an epitope of: the polypeptide sequence shown in SEQ ID NO:Y; a polypeptide sequence encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide sequence encoded by the portion of SEQ ID NO:X as defined in columns 8 and 9 of Table 2; the polypeptide sequence encoded by the portion of SEQ ID NO:B as defined in column 6 of Table 1C or the complement thereto; the polypeptide sequence encoded by the cDNA contained in ATCC Deposit No:Z; or the polypeptide sequence encoded by a polynucleotide that hybridizes to the sequence of SEQ ID NO:X, the complement of the sequence of SEQ ID NO:X, the complement of a portion of SEQ ID NO:X as defined in columns 8 and 9 of Table 2, or the cDNA sequence contained in ATCC Deposit No:Z under stringent hybridization conditions or alternatively, under lower stringency hybridization as defined supra. The present invention further encompasses polynucleotide sequences encoding an epitope of a polypeptide sequence of the invention (such as, for example, the sequence disclosed in SEQ ID NO:X, or a fragment thereof), polynucleotide sequences of the complementary strand of a polynucleotide sequence encoding an epitope of the invention, and polynucleotide sequences which hybridize to the complementary strand under stringent hybridization conditions or alternatively, under lower stringency hybridization conditions defined supra.

The term "epitopes," as used herein, refers to portions of a polypeptide having antigenic or immunogenic activity in an animal, preferably a mammal, and most preferably in a human. In a preferred embodiment, the present invention encompasses a polypeptide comprising an epitope,

as well as the polynucleotide encoding this polypeptide. An "immunogenic epitope," as used herein, is defined as a portion of a protein that elicits an antibody response in an animal, as determined by any method known in the art, for example, by the methods for generating antibodies described *infra*. (See, for example, Geysen et al., Proc. Natl. Acad. Sci. USA 81:3998-4002 (1983)). The term "antigenic epitope," as used herein, is defined as a portion of a protein to which an antibody can immunospecifically bind its antigen as determined by any method well known in the art, for example, by the immunoassays described herein. Immunospecific binding excludes non-specific binding but does not necessarily exclude cross- reactivity with other antigens. Antigenic epitopes need not necessarily be immunogenic.

Fragments which function as epitopes may be produced by any conventional means. (See, e.g., Houghten, R. A., Proc. Natl. Acad. Sci. USA 82:5131-5135 (1985) further described in U.S. Patent No. 4,631,211.)

In the present invention, antigenic epitopes preferably contain a sequence of at least 4, at least 5, at least 6, at least 7, more preferably at least 8, at least 9, at least 10, at least 11, at least 12, at least 13, at least 14, at least 15, at least 20, at least 25, at least 30, at least 40, at least 50, and, most preferably, between about 15 to about 30 amino acids. Preferred polypeptides comprising immunogenic or antigenic epitopes are at least 10, 15, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, or 100 amino acid residues in length. Additional non-exclusive preferred antigenic epitopes include the antigenic epitopes disclosed herein, as well as portions thereof. Antigenic epitopes are useful, for example, to raise antibodies, including monoclonal antibodies, that specifically bind the epitope. Preferred antigenic epitopes include the antigenic epitopes disclosed herein, as well as any combination of two, three, four, five or more of these antigenic epitopes. Antigenic epitopes can be used as the target molecules in immunoassays. (See, for instance, Wilson et al., Cell 37:767-778 (1984); Sutcliffe et al., Science 219:660-666 (1983)).

Non-limiting examples of epitopes of polypeptides that can be used to generate antibodies of the invention include a polypeptide comprising, or alternatively consisting of, at least one, two, three, four, five, six or more of the portion(s) of SEQ ID NO:Y specified in Table 1B. These polypeptide fragments have been determined to bear antigenic epitopes of the proteins of the invention by the analysis of the Jameson-Wolf antigenic index which is included in the DNAStar suite of computer programs. By "comprise" it is intended that a polypeptide contains at least one, two, three, four, five, six or more of the portion(s) of SEQ ID NO:Y shown in Table 1B, but it may contain additional flanking residues on either the amino or carboxyl termini of the recited portion. Such additional flanking sequences are preferably sequences naturally found adjacent to the portion; i.e., contiguous sequence shown in SEQ ID NO:Y. The flanking sequence may, however, be sequences from a heterologous polypeptide, such as from another protein described herein or from a heterologous polypeptide not described herein. In particular embodiments, epitope portions

of a polypeptide of the invention comprise one, two, three, or more of the portions of SEQ ID NO:Y shown in Table 1B.

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Similarly, immunogenic epitopes can be used, for example, to induce antibodies according to methods well known in the art. See, for instance, Sutcliffe et al., supra; Wilson et al., supra; Chow et al., Proc. Natl. Acad. Sci. USA 82:910-914; and Bittle et al., J. Gen. Virol. 66:2347-2354 (1985). Preferred immunogenic epitopes include the immunogenic epitopes disclosed herein, as well as any combination of two, three, four, five or more of these immunogenic epitopes. The polypeptides comprising one or more immunogenic epitopes may be presented for eliciting an antibody response together with a carrier protein, such as an albumin, to an animal system (such as rabbit or mouse), or, if the polypeptide is of sufficient length (at least about 25 amino acids), the polypeptide may be presented without a carrier. However, immunogenic epitopes comprising as few as 8 to 10 amino acids have been shown to be sufficient to raise antibodies capable of binding to, at the very least, linear epitopes in a denatured polypeptide (e.g., in Western blotting).

Epitope-bearing polypeptides of the present invention may be used to induce antibodies according to methods well known in the art including, but not limited to, in vivo immunization, in vitro immunization, and phage display methods. See, e.g., Sutcliffe et al., supra; Wilson et al., supra, and Bittle et al., J. Gen. Virol., 66:2347-2354 (1985). If in vivo immunization is used, animals may be immunized with free peptide; however, anti-peptide antibody titer may be boosted by coupling the peptide to a macromolecular carrier, such as keyhole limpet hemacyanin (KLH) or tetanus toxoid. For instance, peptides containing cysteine residues may be coupled to a carrier using a linker such as maleimidobenzoyl- N-hydroxysuccinimide ester (MBS), while other peptides may be coupled to carriers using a more general linking agent such as glutaraldehyde. Animals such as rabbits, rats and mice are immunized with either free or carrier- coupled peptides, for instance, by intraperitoneal and/or intradermal injection of emulsions containing about 100 µg of peptide or carrier protein and Freund's adjuvant or any other adjuvant known for stimulating an immune response. Several booster injections may be needed, for instance, at intervals of about two weeks, to provide a useful titer of anti-peptide antibody which can be detected, for example, by ELISA assay using free peptide adsorbed to a solid surface. The titer of anti-peptide antibodies in serum from an immunized animal may be increased by selection of anti-peptide antibodies, for instance, by adsorption to the peptide on a solid support and elution of the selected antibodies according to methods well known in the art.

As one of skill in the art will appreciate, and as discussed above, the polypeptides of the present invention (e.g., those comprising an immunogenic or antigenic epitope) can be fused to heterologous polypeptide sequences. For example, polypeptides of the present invention (including fragments or variants thereof), may be fused with the constant domain of immunoglobulins (IgA, IgE, IgG, IgM), or portions thereof (CH1, CH2, CH3, or any combination

thereof and portions thereof, resulting in chimeric polypeptides. By way of another non-limiting example, polypeptides and/or antibodies of the present invention (including fragments or variants thereof) may be fused with albumin (including but not limited to recombinant human serum albumin or fragments or variants thereof (see, e.g., U.S. Patent No. 5,876,969, issued March 2, 1999, EP Patent 0 413 622, and U.S. Patent No. 5,766,883, issued June 16, 1998, herein incorporated by reference in their entirety)). In a preferred embodiment, polypeptides and/or antibodies of the present invention (including fragments or variants thereof) are fused with the mature form of human serum albumin (i.e., amino acids 1 - 585 of human serum albumin as shown in Figures 1 and 2 of EP Patent 0 322 094) which is herein incorporated by reference in its entirety. In another preferred embodiment, polypeptides and/or antibodies of the present invention (including fragments or variants thereof) are fused with polypeptide fragments comprising, or alternatively consisting of, amino acid residues 1-z of human serum albumin, where z is an integer from 369 to 419, as described in U.S. Patent 5,766,883 herein incorporated by reference in its entirety. Polypeptides and/or antibodies of the present invention (including fragments or variants thereof) may be fused to either the N- or C-terminal end of the heterologous protein (e.g., immunoglobulin Fc polypeptide or human serum albumin polypeptide). Polynucleotides encoding fusion proteins of the invention are also encompassed by the invention.

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Such fusion proteins as those described above may facilitate purification and may increase half-life in vivo. This has been shown for chimeric proteins consisting of the first two domains of the human CD4-polypeptide and various domains of the constant regions of the heavy or light chains of mammalian immunoglobulins. See, e.g., EP 394,827; Traunecker et al., Nature, 331:84-86 (1988). Enhanced delivery of an antigen across the epithelial barrier to the immune system has been demonstrated for antigens (e.g., insulin) conjugated to an FcRn binding partner such as IgG or Fc fragments (see, e.g., PCT Publications WO 96/22024 and WO 99/04813). IgG fusion proteins that have a disulfide-linked dimeric structure due to the IgG portion desulfide bonds have also been found to be more efficient in binding and neutralizing other molecules than monomeric polypeptides or fragments thereof alone. See, e.g., Fountoulakis et al., J. Biochem., 270:3958-3964 (1995). Nucleic acids encoding the above epitopes can also be recombined with a gene of interest as an epitope tag (e.g., the hemagglutinin (HA) tag or flag tag) to aid in detection and purification of the expressed polypeptide. For example, a system described by Janknecht et al. allows for the ready purification of non-denatured fusion proteins expressed in human cell lines (Janknecht et al., 1991, Proc. Natl. Acad. Sci. USA 88:8972-897). In this system, the gene of interest is subcloned into a vaccinia recombination plasmid such that the open reading frame of the gene is translationally fused to an amino-terminal tag consisting of six histidine residues. The tag serves as a matrix binding domain for the fusion protein. Extracts from cells infected with the recombinant vaccinia virus are loaded onto Ni2+ nitriloacetic acid-agarose column and histidinetagged proteins can be selectively eluted with imidazole-containing buffers.

#### Fusion Proteins

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Any polypeptide of the present invention can be used to generate fusion proteins. For example, the polypeptide of the present invention, when fused to a second protein, can be used as an antigenic tag. Antibodies raised against the polypeptide of the present invention can be used to indirectly detect the second protein by binding to the polypeptide. Moreover, because secreted proteins target cellular locations based on trafficking signals, polypeptides of the present invention which are shown to be secreted can be used as targeting molecules once fused to other proteins.

Examples of domains that can be fused to polypeptides of the present invention include not only heterologous signal sequences, but also other heterologous functional regions. The fusion does not necessarily need to be direct, but may occur through linker sequences.

In certain preferred embodiments, proteins of the invention are fusion proteins comprising an amino acid sequence that is an N and/or C- terminal deletion of a polypeptide of the invention. In preferred embodiments, the invention is directed to a fusion protein comprising an amino acid sequence that is at least 90%, 95%, 96%, 97%, 98% or 99% identical to a polypeptide sequence of the invention. Polynucleotides encoding these proteins are also encompassed by the invention.

Moreover, fusion proteins may also be engineered to improve characteristics of the polypeptide of the present invention. For instance, a region of additional amino acids, particularly charged amino acids, may be added to the N-terminus of the polypeptide to improve stability and persistence during purification from the host cell or subsequent handling and storage. Also, peptide moieties may be added to the polypeptide to facilitate purification. Such regions may be removed prior to final preparation of the polypeptide. The addition of peptide moieties to facilitate handling of polypeptides are familiar and routine techniques in the art.

As one of skill in the art will appreciate that, as discussed above, polypeptides of the present invention, and epitope-bearing fragments thereof, can be combined with heterologous polypeptide sequences. For example, the polypeptides of the present invention may be fused with heterologous polypeptide sequences, for example, the polypeptides of the present invention may be fused with the constant domain of immunoglobulins (IgA, IgE, IgG, IgM) or portions thereof (CH1, CH2, CH3, and any combination thereof, including both entire domains and portions thereof), or albumin (including, but not limited to, native or recombinant human albumin or fragments or variants thereof (see, e.g., U.S. Patent No. 5,876,969, issued March 2, 1999, EP Patent 0 413 622, and U.S. Patent No. 5,766,883, issued June 16, 1998, herein incorporated by reference in their entirety)), resulting in chimeric polypeptides. For example, EP-A-O 464 533 (Canadian counterpart 2045869) discloses fusion proteins comprising various portions of constant region of immunoglobulin molecules together with another human protein or part thereof. In many cases, the Fc part in a fusion protein is beneficial in therapy and diagnosis, and thus can result in, for example, improved pharmacokinetic properties (EP-A 0232 262). Alternatively,

deleting the Fc part after the fusion protein has been expressed, detected, and purified, would be desired. For example, the Fc portion may hinder therapy and diagnosis if the fusion protein is used as an antigen for immunizations. In drug discovery, for example, human proteins, such as hIL-5, have been fused with Fc portions for the purpose of high-throughput screening assays to identify antagonists of hIL-5. See, D. Bennett et al., J. Molecular Recognition 8:52-58 (1995); K. Johanson et al., J. Biol. Chem. 270:9459-9471 (1995).

Moreover, the polypeptides of the present invention can be fused to marker sequences, such as a polypeptide which facilitates purification of the fused polypeptide. In preferred embodiments, the marker amino acid sequence is a hexa-histidine peptide, such as the tag provided in a pQE vector (QIAGEN, Inc., 9259 Eton Avenue, Chatsworth, CA, 91311), among others, many of which are commercially available. As described in Gentz et al., Proc. Natl. Acad. Sci. USA 86:821-824 (1989), for instance, hexa-histidine provides for convenient purification of the fusion protein. Another peptide tag useful for purification, the "HA" tag, corresponds to an epitope derived from the influenza hemagglutinin protein (Wilson et al., Cell 37:767 (1984)).

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Additional fusion proteins of the invention may be generated through the techniques of gene-shuffling, motif-shuffling, exon-shuffling, and/or codon-shuffling (collectively referred to as "DNA shuffling"). DNA shuffling may be employed to modulate the activities of polypeptides of the invention, such methods can be used to generate polypeptides with altered activity, as well as agonists and antagonists of the polypeptides. See, generally, U.S. Patent Nos. 5,605,793; 5,811,238; 5,830,721; 5,834,252; and 5,837,458, and Patten et al., Curr. Opinion Biotechnol. 8:724-33 (1997); Harayama, Trends Biotechnol. 16(2):76-82 (1998); Hansson, et al., J. Mol. Biol. 287:265-76 (1999); and Lorenzo and Blasco, Biotechniques 24(2):308-13 (1998) (each of these patents and publications are hereby incorporated by reference in its entirety). In one embodiment, alteration of polynucleotides corresponding to SEQ ID NO:X and the polypeptides encoded by these polynucleotides may be achieved by DNA shuffling. DNA shuffling involves the assembly of two or more DNA segments by homologous or site-specific recombination to generate variation in the polynucleotide sequence. In another embodiment, polynucleotides of the invention, or the encoded polypeptides, may be altered by being subjected to random mutagenesis by error-prone PCR, random nucleotide insertion or other methods prior to recombination. In another embodiment, one or more components, motifs, sections, parts, domains, fragments, etc., of a polynucleotide encoding a polypeptide of the invention may be recombined with one or more components, motifs, sections, parts, domains, fragments, etc. of one or more heterologous molecules.

Thus, any of these above fusions can be engineered using the polynucleotides or the polypeptides of the present invention.

Recombinant and Synthetic Production of Polypeptides of the Invention

The present invention also relates to vectors containing the polynucleotide of the present invention, host cells, and the production of polypeptides by synthetic and recombinant techniques. The vector may be, for example, a phage, plasmid, viral, or retroviral vector. Retroviral vectors may be replication competent or replication defective. In the latter case, viral propagation generally will occur only in complementing host cells.

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The polynucleotides of the invention may be joined to a vector containing a selectable marker for propagation in a host. Generally, a plasmid vector is introduced in a precipitate, such as a calcium phosphate precipitate, or in a complex with a charged lipid. If the vector is a virus, it may be packaged in vitro using an appropriate packaging cell line and then transduced into host cells.

The polynucleotide insert should be operatively linked to an appropriate promoter, such as the phage lambda PL promoter, the E. coli lac, trp, phoA and tac promoters, the SV40 early and late promoters and promoters of retroviral LTRs, to name a few. Other suitable promoters will be known to the skilled artisan. The expression constructs will further contain sites for transcription initiation, termination, and, in the transcribed region, a ribosome binding site for translation. The coding portion of the transcripts expressed by the constructs will preferably include a translation initiating codon at the beginning and a termination codon (UAA, UGA or UAG) appropriately positioned at the end of the polypeptide to be translated.

As indicated, the expression vectors will preferably include at least one selectable marker. Such markers include dihydrofolate reductase, G418, glutamine synthase, or neomycin resistance for eukaryotic cell culture, and tetracycline, kanamycin or ampicillin resistance genes for culturing in E. coli and other bacteria. Representative examples of appropriate hosts include, but are not limited to, bacterial cells, such as E. coli, Streptomyces and Salmonella typhimurium cells; fungal cells, such as yeast cells (e.g., Saccharomyces cerevisiae or Pichia pastoris (ATCC Accession No. 201178)); insect cells such as Drosophila S2 and Spodoptera Sf9 cells; animal cells such as CHO, COS, 293, and Bowes melanoma cells; and plant cells. Appropriate culture mediums and conditions for the above-described host cells are known in the art.

Among vectors preferred for use in bacteria include pQE70, pQE60 and pQE-9, available from QIAGEN, Inc.; pBluescript vectors, Phagescript vectors, pNH8A, pNH16a, pNH18A, pNH46A, available from Stratagene Cloning Systems, Inc.; and ptrc99a, pKK223-3, pKK233-3, pDR540, pRIT5 available from Pharmacia Biotech, Inc. Among preferred eukaryotic vectors are pWLNEO, pSV2CAT, pOG44, pXT1 and pSG available from Stratagene; and pSVK3, pBPV, pMSG and pSVL available from Pharmacia. Preferred expression vectors for use in yeast systems include, but are not limited to pYES2, pYD1, pTEF1/Zeo, pYES2/GS, pPICZ, pGAPZ, pGAPZalph, pPIC9, pPIC3.5, pHIL-D2, pHIL-S1, pPIC3.5K, pPIC9K, and PAO815 (all available from Invitrogen, Carlbad, CA). Other suitable vectors will be readily apparent to the skilled artisan.

Vectors which use glutamine synthase (GS) or DHFR as the selectable markers can be amplified in the presence of the drugs methionine sulphoximine or methotrexate, respectively. An advantage of glutamine synthase based vectors are the availability of cell lines (e.g., the murine myeloma cell line, NS0) which are glutamine synthase negative. Glutamine synthase expression systems can also function in glutamine synthase expressing cells (e.g., Chinese Hamster Ovary (CHO) cells) by providing additional inhibitor to prevent the functioning of the endogenous gene. A glutamine synthase expression system and components thereof are detailed in PCT publications: WO87/04462; WO86/05807; WO89/01036; WO89/10404; and WO91/06657, which are hereby incorporated in their entireties by reference herein. Additionally, glutamine synthase expression vectors can be obtained from Lonza Biologics, Inc. (Portsmouth, NH). Expression and production of monoclonal antibodies using a GS expression system in murine myeloma cells is described in Bebbington et al., Bio/technology 10:169(1992) and in Biblia and Robinson Biotechnol. Prog. 11:1 (1995) which are herein incorporated by reference.

The present invention also relates to host cells containing the above-described vector constructs described herein, and additionally encompasses host cells containing nucleotide sequences of the invention that are operably associated with one or more heterologous control regions (e.g., promoter and/or enhancer) using techniques known of in the art. The host cell can be a higher eukaryotic cell, such as a mammalian cell (e.g., a human derived cell), or a lower eukaryotic cell, such as a yeast cell, or the host cell can be a prokaryotic cell, such as a bacterial cell. A host strain may be chosen which modulates the expression of the inserted gene sequences, or modifies and processes the gene product in the specific fashion desired. Expression from certain promoters can be elevated in the presence of certain inducers; thus expression of the genetically engineered polypeptide may be controlled. Furthermore, different host cells have characteristics and specific mechanisms for the translational and post-translational processing and modification (e.g., phosphorylation, cleavage) of proteins. Appropriate cell lines can be chosen to ensure the desired modifications and processing of the foreign protein expressed.

Introduction of the nucleic acids and nucleic acid constructs of the invention into the host cell can be effected by calcium phosphate transfection, DEAE-dextran mediated transfection, cationic lipid-mediated transfection, electroporation, transduction, infection, or other methods. Such methods are described in many standard laboratory manuals, such as Davis et al., Basic Methods In Molecular Biology (1986). It is specifically contemplated that the polypeptides of the present invention may in fact be expressed by a host cell lacking a recombinant vector.

In addition to encompassing host cells containing the vector constructs discussed herein, the invention also encompasses primary, secondary, and immortalized host cells of vertebrate origin, particularly mammalian origin, that have been engineered to delete or replace endogenous genetic material (e.g., the coding sequence), and/or to include genetic material (e.g., heterologous polynucleotide sequences) that is operably associated with polynucleotides of the invention, and

which activates, alters, and/or amplifies endogenous polynucleotides. For example, techniques known in the art may be used to operably associate heterologous control regions (e.g., promoter and/or enhancer) and endogenous polynucleotide sequences via homologous recombination (see, e.g., US Patent Number 5,641,670, issued June 24, 1997; International Publication Number WO 96/29411; International Publication Number WO 94/12650; Koller et al., Proc. Natl. Acad. Sci. USA 86:8932-8935 (1989); and Zijlstra et al., Nature 342:435-438 (1989), the disclosures of each of which are incorporated by reference in their entireties).

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Polypeptides of the invention can be recovered and purified from recombinant cell cultures by well-known methods including ammonium sulfate or ethanol precipitation, acid extraction, anion or cation exchange chromatography, phosphocellulose chromatography, hydrophobic interaction chromatography, affinity chromatography, hydroxylapatite chromatography and lectin chromatography. Most preferably, high performance liquid chromatography ("HPLC") is employed for purification.

Polypeptides of the present invention can also be recovered from: products purified from natural sources, including bodily fluids, tissues and cells, whether directly isolated or cultured; products of chemical synthetic procedures; and products produced by recombinant techniques from a prokaryotic or eukaryotic host, including, for example, bacterial, yeast, higher plant, insect, and mammalian cells. Depending upon the host employed in a recombinant production procedure, the polypeptides of the present invention may be glycosylated or may be non-glycosylated. In addition, polypeptides of the invention may also include an initial modified methionine residue, in some cases as a result of host-mediated processes. Thus, it is well known in the art that the N-terminal methionine encoded by the translation initiation codon generally is removed with high efficiency from any protein after translation in all eukaryotic cells. While the N-terminal methionine on most proteins also is efficiently removed in most prokaryotes, for some proteins, this prokaryotic removal process is inefficient, depending on the nature of the amino acid to which the N-terminal methionine is covalently linked.

In one embodiment, the yeast *Pichia pastoris* is used to express polypeptides of the invention in a eukaryotic system. *Pichia pastoris* is a methylotrophic yeast which can metabolize methanol as its sole carbon source. A main step in the methanol metabolization pathway is the oxidation of methanol to formaldehyde using O₂. This reaction is catalyzed by the enzyme alcohol oxidase. In order to metabolize methanol as its sole carbon source, *Pichia pastoris* must generate high levels of alcohol oxidase due, in part, to the relatively low affinity of alcohol oxidase for O₂. Consequently, in a growth medium depending on methanol as a main carbon source, the promoter region of one of the two alcohol oxidase genes (*AOXI*) is highly active. In the presence of methanol, alcohol oxidase produced from the *AOXI* gene comprises up to approximately 30% of the total soluble protein in *Pichia pastoris*. See Ellis, S.B., et al., Mol. Cell. Biol. 5:1111-21

(1985); Koutz, P.J, et al., Yeast 5:167-77 (1989); Tschopp, J.F., et al., Nucl. Acids Res. 15:3859-76 (1987). Thus, a heterologous coding sequence, such as, for example, a polynucleotide of the present invention, under the transcriptional regulation of all or part of the AOX1 regulatory sequence is expressed at exceptionally high levels in Pichia yeast grown in the presence of methanol.

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In one example, the plasmid vector pPIC9K is used to express DNA encoding a polypeptide of the invention, as set forth herein, in a *Pichea* yeast system essentially as described in "*Pichia* Protocols: Methods in Molecular Biology," D.R. Higgins and J. Cregg, eds. The Humana Press, Totowa, NJ, 1998. This expression vector allows expression and secretion of a polypeptide of the invention by virtue of the strong *AOX1* promoter linked to the *Pichia pastoris* alkaline phosphatase (PHO) secretory signal peptide (i.e., leader) located upstream of a multiple cloning site.

Many other yeast vectors could be used in place of pPIC9K, such as, pYES2, pYD1, pTEF1/Zeo, pYES2/GS, pPICZ, pGAPZ, pGAPZalpha, pPIC9, pPIC3.5, pHIL-D2, pHIL-S1, pPIC3.5K, and PAO815, as one skilled in the art would readily appreciate, as long as the proposed expression construct provides appropriately located signals for transcription, translation, secretion (if desired), and the like, including an in-frame AUG as required.

In another embodiment, high-level expression of a heterologous coding sequence, such as, for example, a polynucleotide of the present invention, may be achieved by cloning the heterologous polynucleotide of the invention into an expression vector such as, for example, pGAPZ or pGAPZalpha, and growing the yeast culture in the absence of methanol.

In addition to encompassing host cells containing the vector constructs discussed herein, the invention also encompasses primary, secondary, and immortalized host cells of vertebrate origin, particularly mammalian origin, that have been engineered to delete or replace endogenous genetic material (e.g., coding sequence), and/or to include genetic material (e.g., heterologous polynucleotide sequences) that is operably associated with polynucleotides of the invention, and which activates, alters, and/or amplifies endogenous polynucleotides. For example, techniques known in the art may be used to operably associate heterologous control regions (e.g., promoter and/or enhancer) and endogenous polynucleotide sequences via homologous recombination (see, e.g., U.S. Patent No. 5,641,670, issued June 24, 1997; International Publication No. WO 96/29411, published September 26, 1996; International Publication No. WO 94/12650, published August 4, 1994; Koller et al., Proc. Natl. Acad. Sci. USA 86:8932-8935 (1989); and Zijlstra et al., Nature 342:435-438 (1989), the disclosures of each of which are incorporated by reference in their entireties).

In addition, polypeptides of the invention can be chemically synthesized using techniques

known in the art (e.g., see Creighton, 1983, Proteins: Structures and Molecular Principles, W.H. Freeman & Co., N.Y., and Hunkapiller et al., *Nature*, 310:105-111 (1984)). For example, a polypeptide corresponding to a fragment of a polypeptide can be synthesized by use of a peptide synthesizer. Furthermore, if desired, nonclassical amino acids or chemical amino acid analogs can be introduced as a substitution or addition into the polypeptide sequence. Non-classical amino acids include, but are not limited to, to the D-isomers of the common amino acids, 2,4-diaminobutyric acid, a-amino isobutyric acid, 4-aminobutyric acid, Abu, 2-amino butyric acid, g-Abu, e-Ahx, 6-amino hexanoic acid, Aib, 2-amino isobutyric acid, 3-amino propionic acid, ornithine, norleucine, norvaline, hydroxyproline, sarcosine, citrulline, homocitrulline, cysteic acid, t-butylglycine, t-butylalanine, phenylglycine, cyclohexylalanine, b-alanine, fluoro-amino acids, designer amino acids such as b-methyl amino acids, Ca-methyl amino acids, Na-methyl amino acids, and amino acid analogs in general. Furthermore, the amino acid can be D (dextrorotary) or L (levorotary).

The invention encompasses polypeptides of the present invention which are differentially modified during or after translation, e.g., by glycosylation, acetylation, phosphorylation, amidation, derivatization by known protecting/blocking groups, proteolytic cleavage, linkage to an antibody molecule or other cellular ligand, etc. Any of numerous chemical modifications may be carried out by known techniques, including but not limited, to specific chemical cleavage by cyanogen bromide, trypsin, chymotrypsin, papain, V8 protease, NaBH₄; acetylation, formylation, oxidation, reduction; metabolic synthesis in the presence of tunicamycin; etc.

Additional post-translational modifications encompassed by the invention include, for example, e.g., N-linked or O-linked carbohydrate chains, processing of N-terminal or C-terminal ends), attachment of chemical moieties to the amino acid backbone, chemical modifications of N-linked or O-linked carbohydrate chains, and addition or deletion of an N-terminal methionine residue as a result of procaryotic host cell expression. The polypeptides may also be modified with a detectable label, such as an enzymatic, fluorescent, isotopic or affinity label to allow for detection and isolation of the protein.

Examples of suitable enzymes include horseradish peroxidase, alkaline phosphatase, beta-galactosidase, or acetylcholinesterase; examples of suitable prosthetic group complexes include streptavidin/biotin and avidin/biotin; examples of suitable fluorescent materials include umbelliferone, fluorescein, fluorescein isothiocyanate, rhodamine, dichlorotriazinylamine fluorescein, dansyl chloride or phycoerythrin; an example of a luminescent material includes luminol; examples of bioluminescent materials include luciferase, luciferin, and aequorin; and examples of suitable radioactive material include iodine (121 I, 123 I, 125 I, 131 I), carbon (14 C), sulfur (35 S), tritium (3H), indium (111 In, 112 In, 113m In, 115m In), technetium (99 Tc, 99m Tc), thallium (201 Ti), gallium (68 Ga, 67 Ga), palladium (103 Pd), molybdenum (99 Mo), xenon (133 Xe), fluorine (18 F), 153 Sm, 177 Lu, 159 Gd, 149 Pm, 140 La, 175 Yb, 166 Ho, 90 Y, 47 Sc, 186 Re, 188 Re, 142 Pr, 105 Rh, and 97 Ru.

In specific embodiments, a polypeptide of the present invention or fragment or variant thereof is attached to macrocyclic chelators that associate with radiometal ions, including but not limited to, ¹⁷⁷Lu, ⁹⁰Y, ¹⁶⁶Ho, and ¹⁵³Sm, to polypeptides. In a preferred embodiment, the radiometal ion associated with the macrocyclic chelators is ¹¹¹In. In another preferred embodiment, the radiometal ion associated with the macrocyclic chelator is ⁹⁰Y. In specific embodiments, the macrocyclic chelator is 1,4,7,10-tetraazacyclododecane-N,N',N'',N'''-tetraacetic acid (DOTA). In other specific embodiments, DOTA is attached to an antibody of the invention or fragment thereof via a linker molecule. Examples of linker molecules useful for conjugating DOTA to a polypeptide are commonly known in the art - see, for example, DeNardo et al., Clin Cancer Res. 4(10):2483-90 (1998); Peterson et al., Bioconjug. Chem. 10(4):553-7 (1999); and Zimmerman et al, Nucl. Med. Biol. 26(8):943-50 (1999); which are hereby incorporated by reference in their entirety.

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As mentioned, the proteins of the invention may be modified by either natural processes, such as posttranslational processing, or by chemical modification techniques which are well known in the art. It will be appreciated that the same type of modification may be present in the same or varying degrees at several sites in a given polypeptide. Polypeptides of the invention may be branched, for example, as a result of ubiquitination, and they may be cyclic, with or without branching. Cyclic, branched, and branched cyclic polypeptides may result from posttranslation natural processes or may be made by synthetic methods. Modifications include acetylation, acylation, ADP-ribosylation, amidation, covalent attachment of flavin, covalent attachment of a heme moiety, covalent attachment of a nucleotide or nucleotide derivative, covalent attachment of a lipid or lipid derivative, covalent attachment of phosphotidylinositol, cross-linking, cyclization, disulfide bond formation, demethylation, formation of covalent cross-links, formation of cysteine, formation of pyroglutamate, formylation, gamma-carboxylation, glycosylation, GPI anchor formation, hydroxylation, iodination, methylation, myristoylation, oxidation, pegylation, proteolytic processing, phosphorylation, prenylation, racemization, selenoylation, sulfation, transfer-RNA mediated addition of amino acids to proteins such as arginylation, and ubiquitination. (See, for instance, PROTEINS - STRUCTURE AND MOLECULAR PROPERTIES, 2nd Ed., T. E. Creighton, W. H. Freeman and Company, New York (1993); POSTTRANSLATIONAL COVALENT MODIFICATION OF PROTEINS, B. C. Johnson, Ed., Academic Press, New York, pgs. 1-12 (1983); Seifter et al., Meth. Enzymol. 182:626-646 (1990); Rattan et al., Ann. N.Y. Acad. Sci. 663:48-62 (1992)).

Also provided by the invention are chemically modified derivatives of the polypeptides of the invention which may provide additional advantages such as increased solubility, stability and circulating time of the polypeptide, or decreased immunogenicity (see U.S. Patent No. 4,179,337). The chemical moieties for derivitization may be selected from water soluble polymers such as polyethylene glycol, ethylene glycol/propylene glycol copolymers, carboxymethylcellulose,

dextran, polyvinyl alcohol and the like. The polypeptides may be modified at random positions within the molecule, or at predetermined positions within the molecule and may include one, two, three or more attached chemical moieties.

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The polymer may be of any molecular weight, and may be branched or unbranched. For polyethylene glycol, the preferred molecular weight is between about 1 kDa and about 100 kDa (the term "about" indicating that in preparations of polyethylene glycol, some molecules will weigh more, some less, than the stated molecular weight) for ease in handling and manufacturing. Other sizes may be used, depending on the desired therapeutic profile (e.g., the duration of sustained release desired, the effects, if any on biological activity, the ease in handling, the degree or lack of antigenicity and other known effects of the polyethylene glycol to a therapeutic protein or analog). For example, the polyethylene glycol may have an average molecular weight of about 200, 500, 1000, 1500, 2000, 2500, 3000, 3500, 4000, 4500, 5000, 5500, 6000, 6500, 7000, 7500, 8000, 8500, 9000, 9500, 10,000, 10,500, 11,000, 11,500, 12,000, 12,500, 13,000, 13,500, 14,000, 14,500, 15,000, 15,500, 16,000, 16,500, 17,000, 17,500, 18,000, 18,500, 19,000, 19,500, 20,000, 25,000, 30,000, 35,000, 40,000, 45,000, 50,000, 55,000, 60,000, 65,000, 70,000, 75,000, 80,000, 85,000, 90,000, 95,000, or 100,000 kDa.

As noted above, the polyethylene glycol may have a branched structure. Branched polyethylene glycols are described, for example, in U.S. Patent No. 5,643,575; Morpurgo et al., Appl. Biochem. Biotechnol. 56:59-72 (1996); Vorobjev et al., Nucleosides Nucleotides 18:2745-2750 (1999); and Caliceti et al., Bioconjug. Chem. 10:638-646 (1999), the disclosures of each of which are incorporated herein by reference.

The polyethylene glycol molecules (or other chemical moieties) should be attached to the protein with consideration of effects on functional or antigenic domains of the protein. There are a number of attachment methods available to those skilled in the art, such as, for example, the method disclosed in EP 0 401 384 (coupling PEG to G-CSF), herein incorporated by reference; see also Malik et al., Exp. Hematol. 20:1028-1035 (1992), reporting pegylation of GM-CSF using tresyl chloride. For example, polyethylene glycol may be covalently bound through amino acid residues via a reactive group, such as a free amino or carboxyl group. Reactive groups are those to which an activated polyethylene glycol molecule may be bound. The amino acid residues having a free amino group may include lysine residues and the N-terminal amino acid residues; those having a free carboxyl group may include aspartic acid residues glutamic acid residues and the C-terminal amino acid residue. Sulfhydryl groups may also be used as a reactive group for attaching the polyethylene glycol molecules. Preferred for therapeutic purposes is attachment at an amino group, such as attachment at the N-terminus or lysine group.

As suggested above, polyethylene glycol may be attached to proteins via linkage to any of a number of amino acid residues. For example, polyethylene glycol can be linked to proteins via covalent bonds to lysine, histidine, aspartic acid, glutamic acid, or cysteine residues. One or more

reaction chemistries may be employed to attach polyethylene glycol to specific amino acid residues (e.g., lysine, histidine, aspartic acid, glutamic acid, or cysteine) of the protein or to more than one type of amino acid residue (e.g., lysine, histidine, aspartic acid, glutamic acid, cysteine and combinations thereof) of the protein.

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One may specifically desire proteins chemically modified at the N-terminus. Using polyethylene glycol as an illustration of the present composition, one may select from a variety of polyethylene glycol molecules (by molecular weight, branching, etc.), the proportion of polyethylene glycol molecules to protein (polypeptide) molecules in the reaction mix, the type of pegylation reaction to be performed, and the method of obtaining the selected N-terminally pegylated protein. The method of obtaining the N-terminally pegylated preparation (i.e., separating this moiety from other monopegylated moieties if necessary) may be by purification of the N-terminally pegylated material from a population of pegylated protein molecules. Selective proteins chemically modified at the N-terminus modification may be accomplished by reductive alkylation which exploits differential reactivity of different types of primary amino groups (lysine versus the N-terminal) available for derivatization in a particular protein. Under the appropriate reaction conditions, substantially selective derivatization of the protein at the N-terminus with a carbonyl group containing polymer is achieved.

As indicated above, pegylation of the proteins of the invention may be accomplished by any number of means. For example, polyethylene glycol may be attached to the protein either directly or by an intervening linker. Linkerless systems for attaching polyethylene glycol to proteins are described in Delgado et al., Crit. Rev. Thera. Drug Carrier Sys. 9:249-304 (1992); Francis et al., Intern. J. of Hematol. 68:1-18 (1998); U.S. Patent No. 4,002,531; U.S. Patent No. 5,349,052; WO 95/06058; and WO 98/32466, the disclosures of each of which are incorporated herein by reference.

One system for attaching polyethylene glycol directly to amino acid residues of proteins without an intervening linker employs tresylated MPEG, which is produced by the modification of monmethoxy polyethylene glycol (MPEG) using tresylchloride (ClSO₂CH₂CF₃). Upon reaction of protein with tresylated MPEG, polyethylene glycol is directly attached to amine groups of the protein. Thus, the invention includes protein-polyethylene glycol conjugates produced by reacting proteins of the invention with a polyethylene glycol molecule having a 2,2,2-trifluoreothane sulphonyl group.

Polyethylene glycol can also be attached to proteins using a number of different intervening linkers. For example, U.S. Patent No. 5,612,460, the entire disclosure of which is incorporated herein by reference, discloses urethane linkers for connecting polyethylene glycol to proteins. Protein-polyethylene glycol conjugates wherein the polyethylene glycol is attached to the protein by a linker can also be produced by reaction of proteins with compounds such as MPEG-succinimidylsuccinate, MPEG activated with 1,1'-carbonyldiimidazole, MPEG-

2,4,5-trichloropenylcarbonate, MPEG-p-nitrophenolcarbonate, and various MPEG-succinate derivatives. A number of additional polyethylene glycol derivatives and reaction chemistries for attaching polyethylene glycol to proteins are described in International Publication No. WO 98/32466, the entire disclosure of which is incorporated herein by reference. Pegylated protein products produced using the reaction chemistries set out herein are included within the scope of the invention.

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The number of polyethylene glycol moieties attached to each protein of the invention (i.e., the degree of substitution) may also vary. For example, the pegylated proteins of the invention may be linked, on average, to 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 12, 15, 17, 20, or more polyethylene glycol molecules. Similarly, the average degree of substitution within ranges such as 1-3, 2-4, 3-5, 4-6, 5-7, 6-8, 7-9, 8-10, 9-11, 10-12, 11-13, 12-14, 13-15, 14-16, 15-17, 16-18, 17-19, or 18-20 polyethylene glycol moieties per protein molecule. Methods for determining the degree of substitution are discussed, for example, in Delgado et al., Crit. Rev. Thera. Drug Carrier Sys. 9:249-304 (1992).

The polypeptides of the invention can be recovered and purified from chemical synthesis and recombinant cell cultures by standard methods which include, but are not limited to, ammonium sulfate or ethanol precipitation, acid extraction, anion or cation exchange chromatography, phosphocellulose chromatography, hydrophobic interaction chromatography, affinity chromatography, hydroxylapatite chromatography and lectin chromatography. Most preferably, high performance liquid chromatography ("HPLC") is employed for purification. Well known techniques for refolding protein may be employed to regenerate active conformation when the polypeptide is denatured during isolation and/or purification.

The polypeptides of the invention may be in monomers or multimers (i.e., dimers, trimers, tetramers and higher multimers). Accordingly, the present invention relates to monomers and multimers of the polypeptides of the invention, their preparation, and compositions (preferably, Therapeutics) containing them. In specific embodiments, the polypeptides of the invention are monomers, dimers, trimers or tetramers. In additional embodiments, the multimers of the invention are at least dimers, at least trimers, or at least tetramers.

Multimers encompassed by the invention may be homomers or heteromers. As used herein, the term homomer refers to a multimer containing only polypeptides corresponding to a protein of the invention (e.g., the amino acid sequence of SEQ ID NO:Y, an amino acid sequence encoded by SEQ ID NO:X or the complement of SEQ ID NO:X, the amino acid sequence encoded by the portion of SEQ ID NO:X as defined in columns 8 and 9 of Table 2, and/or an amino acid sequence encoded by cDNA contained in ATCC Deposit No:Z (including fragments, variants, splice variants, and fusion proteins, corresponding to these as described herein)). These homomers may contain polypeptides having identical or different amino acid sequences. In a specific embodiment, a homomer of the invention is a multimer containing only polypeptides having an

identical amino acid sequence. In another specific embodiment, a homomer of the invention is a multimer containing polypeptides having different amino acid sequences. In specific embodiments, the multimer of the invention is a homodimer (e.g., containing two polypeptides having identical or different amino acid sequences) or a homotrimer (e.g., containing three polypeptides having identical and/or different amino acid sequences). In additional embodiments, the homomeric multimer of the invention is at least a homodimer, at least a homotrimer, or at least a homotetramer.

As used herein, the term heteromer refers to a multimer containing one or more heterologous polypeptides (i.e., polypeptides of different proteins) in addition to the polypeptides of the invention. In a specific embodiment, the multimer of the invention is a heterodimer, a heterotrimer, or a heterotetramer. In additional embodiments, the heteromeric multimer of the invention is at least a heterodimer, at least a heterotrimer, or at least a heterotetramer.

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Multimers of the invention may be the result of hydrophobic, hydrophilic, ionic and/or covalent associations and/or may be indirectly linked by, for example, liposome formation. Thus, in one embodiment, multimers of the invention, such as, for example, homodimers or homotrimers, are formed when polypeptides of the invention contact one another in solution. In another embodiment, heteromultimers of the invention, such as, for example, heterotrimers or heterotetramers, are formed when polypeptides of the invention contact antibodies to the polypeptides of the invention (including antibodies to the heterologous polypeptide sequence in a fusion protein of the invention) in solution. In other embodiments, multimers of the invention are formed by covalent associations with and/or between the polypeptides of the invention. Such covalent associations may involve one or more amino acid residues contained in the polypeptide sequence (e.g., that recited in SEQ ID NO:Y, encoded by the portion of SEQ ID NO:X as defined in columns 8 and 9 of Table 2, and/or encoded by the cDNA contained in ATCC Deposit No:Z). In one instance, the covalent associations are cross-linking between cysteine residues located within the polypeptide sequences which interact in the native (i.e., naturally occurring) polypeptide. In another instance, the covalent associations are the consequence of chemical or recombinant manipulation. Alternatively, such covalent associations may involve one or more amino acid residues contained in the heterologous polypeptide sequence in a fusion protein. In one example, covalent associations are between the heterologous sequence contained in a fusion protein of the invention (see, e.g., US Patent Number 5,478,925). In a specific example, the covalent associations are between the heterologous sequence contained in a Fc fusion protein of the invention (as described herein). In another specific example, covalent associations of fusion proteins of the invention are between heterologous polypeptide sequence from another protein that is capable of forming covalently associated multimers, such as for example, osteoprotegerin (see, e.g., International Publication NO: WO 98/49305, the contents of which are herein incorporated by reference in its entirety). In another embodiment, two or more polypeptides of the invention are

joined through peptide linkers. Examples include those peptide linkers described in U.S. Pat. No. 5,073,627 (hereby incorporated by reference). Proteins comprising multiple polypeptides of the invention separated by peptide linkers may be produced using conventional recombinant DNA technology.

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Another method for preparing multimer polypeptides of the invention involves use of polypeptides of the invention fused to a leucine zipper or isoleucine zipper polypeptide sequence. Leucine zipper and isoleucine zipper domains are polypeptides that promote multimerization of the proteins in which they are found. Leucine zippers were originally identified in several DNA-binding proteins (Landschulz et al., Science 240:1759, (1988)), and have since been found in a variety of different proteins. Among the known leucine zippers are naturally occurring peptides and derivatives thereof that dimerize or trimerize. Examples of leucine zipper domains suitable for producing soluble multimeric proteins of the invention are those described in PCT application WO 94/10308, hereby incorporated by reference. Recombinant fusion proteins comprising a polypeptide of the invention fused to a polypeptide sequence that dimerizes or trimerizes in solution are expressed in suitable host cells, and the resulting soluble multimeric fusion protein is recovered from the culture supernatant using techniques known in the art.

Trimeric polypeptides of the invention may offer the advantage of enhanced biological activity. Preferred leucine zipper moieties and isoleucine moieties are those that preferentially form trimers. One example is a leucine zipper derived from lung surfactant protein D (SPD), as described in Hoppe et al. (FEBS Letters 344:191, (1994)) and in U.S. patent application Ser. No. 08/446,922, hereby incorporated by reference. Other peptides derived from naturally occurring trimeric proteins may be employed in preparing trimeric polypeptides of the invention.

In another example, proteins of the invention are associated by interactions between Flag® polypeptide sequence contained in fusion proteins of the invention containing Flag® polypeptide sequence. In a further embodiment, proteins of the invention are associated by interactions between heterologous polypeptide sequence contained in Flag® fusion proteins of the invention and anti-Flag® antibody.

The multimers of the invention may be generated using chemical techniques known in the art. For example, polypeptides desired to be contained in the multimers of the invention may be chemically cross-linked using linker molecules and linker molecule length optimization techniques known in the art (see, e.g., US Patent Number 5,478,925, which is herein incorporated by reference in its entirety). Additionally, multimers of the invention may be generated using techniques known in the art to form one or more inter-molecule cross-links between the cysteine residues located within the sequence of the polypeptides desired to be contained in the multimer (see, e.g., US Patent Number 5,478,925, which is herein incorporated by reference in its entirety). Further, polypeptides of the invention may be routinely modified by the addition of cysteine or biotin to the C-terminus or N-terminus of the polypeptide and techniques known in the art may be

applied to generate multimers containing one or more of these modified polypeptides (see, e.g., US Patent Number 5,478,925, which is herein incorporated by reference in its entirety). Additionally, techniques known in the art may be applied to generate liposomes containing the polypeptide components desired to be contained in the multimer of the invention (see, e.g., US Patent Number 5,478,925, which is herein incorporated by reference in its entirety).

Alternatively, multimers of the invention may be generated using genetic engineering techniques known in the art. In one embodiment, polypeptides contained in multimers of the invention are produced recombinantly using fusion protein technology described herein or otherwise known in the art (see, e.g., US Patent Number 5,478,925, which is herein incorporated by reference in its entirety). In a specific embodiment, polynucleotides coding for a homodimer of the invention are generated by ligating a polynucleotide sequence encoding a polypeptide of the invention to a sequence encoding a linker polypeptide and then further to a synthetic polynucleotide encoding the translated product of the polypeptide in the reverse orientation from the original C-terminus to the N-terminus (lacking the leader sequence) (see, e.g., US Patent Number 5,478,925, which is herein incorporated by reference in its entirety). In another embodiment, recombinant techniques described herein or otherwise known in the art are applied to generate recombinant polypeptides of the invention which contain a transmembrane domain (or hydrophobic or signal peptide) and which can be incorporated by membrane reconstitution techniques into liposomes (see, e.g., US Patent Number 5,478,925, which is herein incorporated by reference in its entirety).

# **Antibodies**

Further polypeptides of the invention relate to antibodies and T-cell antigen receptors (TCR) which immunospecifically bind a polypeptide, polypeptide fragment, or variant of the invention (e.g., a polypeptide or fragment or variant of the amino acid sequence of SEQ ID NO:Y or a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z, and/or an epitope, of the present invention) as determined by immunoassays well known in the art for assaying specific antibody-antigen binding. Antibodies of the invention include, but are not limited to, polyclonal, monoclonal, multispecific, human, humanized or chimeric antibodies, single chain antibodies, Fab fragments, F(ab') fragments, fragments produced by a Fab expression library, anti-idiotypic (anti-Id) antibodies (including, e.g., anti-Id antibodies to antibodies of the invention), intracellularly-made antibodies (i.e., intrabodies), and epitope-binding fragments of any of the above. The term "antibody," as used herein, refers to immunoglobulin molecules and immunologically active portions of immunoglobulin molecules, i.e., molecules that contain an antigen binding site that immunospecifically binds an antigen. The immunoglobulin molecules of the invention can be of any type (e.g., IgG, IgE, IgM, IgD, IgA and IgY), class (e.g., IgG1, IgG2, IgG3, IgG4, IgA1 and IgA2) or subclass of immunoglobulin molecule. In preferred embodiments, the immunoglobulin

molecules of the invention are IgG1. In other preferred embodiments, the immunoglobulin molecules of the invention are IgG4.

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Most preferably the antibodies are human antigen-binding antibody fragments of the present invention and include, but are not limited to, Fab, Fab' and F(ab')2, Fd, single-chain Fvs (scFv), single-chain antibodies, disulfide-linked Fvs (sdFv) and fragments comprising either a VL or VH domain. Antigen-binding antibody fragments, including single-chain antibodies, may comprise the variable region(s) alone or in combination with the entirety or a portion of the following: hinge region, CH1, CH2, and CH3 domains. Also included in the invention are antigen-binding fragments also comprising any combination of variable region(s) with a hinge region, CH1, CH2, and CH3 domains. The antibodies of the invention may be from any animal origin including birds and mammals. Preferably, the antibodies are human, murine (e.g., mouse and rat), donkey, ship rabbit, goat, guinea pig, camel, horse, or chicken. As used herein, "human" antibodies include antibodies having the amino acid sequence of a human immunoglobulin and include antibodies isolated from human immunoglobulin libraries or from animals transgenic for one or more human immunoglobulin and that do not express endogenous immunoglobulins, as described infra and, for example in, U.S. Patent No. 5,939,598 by Kucherlapati et al.

The antibodies of the present invention may be monospecific, bispecific, trispecific or of greater multispecificity. Multispecific antibodies may be specific for different epitopes of a polypeptide of the present invention or may be specific for both a polypeptide of the present invention as well as for a heterologous epitope, such as a heterologous polypeptide or solid support material. See, e.g., PCT publications WO 93/17715; WO 92/08802; WO 91/00360; WO 92/05793; Tutt, et al., J. Immunol. 147:60-69 (1991); U.S. Patent Nos. 4,474,893; 4,714,681; 4,925,648; 5,573,920; 5,601,819; Kostelny et al., J. Immunol. 148:1547-1553 (1992).

Antibodies of the present invention may be described or specified in terms of the epitope(s) or portion(s) of a polypeptide of the present invention which they recognize or specifically bind. The epitope(s) or polypeptide portion(s) may be specified as described herein, e.g., by N-terminal and C-terminal positions, or by size in contiguous amino acid residues, or listed in the Tables and Figures. Preferred epitopes of the invention include the predicted epitopes shown in Table 1B, as well as polynucleotides that encode these epitopes. Antibodies which specifically bind any epitope or polypeptide of the present invention may also be excluded. Therefore, the present invention includes antibodies that specifically bind polypeptides of the present invention, and allows for the exclusion of the same.

Antibodies of the present invention may also be described or specified in terms of their cross-reactivity. Antibodies that do not bind any other analog, ortholog, or homolog of a polypeptide of the present invention are included. Antibodies that bind polypeptides with at least 95%, at least 90%, at least 85%, at least 80%, at least 75%, at least 70%, at least 65%, at least 60% at least 55%, and at least 50% identity (as calculated using methods known in the art and

described herein) to a polypeptide of the present invention are also included in the present invention. In specific embodiments, antibodies of the present invention cross-react with murine, rat and/or rabbit homologs of human proteins and the corresponding epitopes thereof. Antibodies that do not bind polypeptides with less than 95%, less than 90%, less than 85%, less than 80%, less than 75%, less than 70%, less than 65%, less than 60%, less than 55%, and less than 50% identity (as calculated using methods known in the art and described herein) to a polypeptide of the present invention are also included in the present invention. In a specific embodiment, the abovedescribed cross-reactivity is with respect to any single specific antigenic or immunogenic polypeptide, or combination(s) of 2, 3, 4, 5, or more of the specific antigenic and/or immunogenic polypeptides disclosed herein. Further included in the present invention are antibodies which bind polypeptides encoded by polynucleotides which hybridize to a polynucleotide of the present invention under stringent hybridization conditions (as described herein). Antibodies of the present invention may also be described or specified in terms of their binding affinity to a polypeptide of the invention. Preferred binding affinities include those with a dissociation constant or Kd less than 5 X 10⁻² M, 10⁻² M, 5 X 10⁻³ M, 10⁻³ M, 5 X 10⁻⁴ M, 10⁻⁴ M, 5 X 10⁻⁵ M, 10⁻⁵ M, 5 X 10⁻⁶ M,  $10^{-6}$ M, 5 X  $10^{-7}$  M,  $10^{7}$  M, 5 X  $10^{-8}$  M,  $10^{-8}$  M, 5 X  $10^{-9}$  M,  $10^{-9}$  M, 5 X  $10^{-10}$  M,  $10^{-10}$  M, 5 X  $10^{-11}$ M,  $10^{-11}$  M, 5 X  $10^{-12}$  M,  $10^{-12}$  M, 5 X  $10^{-13}$  M,  $10^{-13}$  M,  $10^{-13}$  M,  $10^{-14}$  M,  $10^{-14}$  M,  $10^{-14}$  M,  $10^{-15}$  M, or  $10^{-15}$ M.

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The invention also provides antibodies that competitively inhibit binding of an antibody to an epitope of the invention as determined by any method known in the art for determining competitive binding, for example, the immunoassays described herein. In preferred embodiments, the antibody competitively inhibits binding to the epitope by at least 95%, at least 90%, at least 85%, at least 75%, at least 70%, at least 60%, or at least 50%.

Antibodies of the present invention may act as agonists or antagonists of the polypeptides of the present invention. For example, the present invention includes antibodies which disrupt the receptor/ligand interactions with the polypeptides of the invention either partially or fully. Preferably, antibodies of the present invention bind an antigenic epitope disclosed herein, or a portion thereof. The invention features both receptor-specific antibodies and ligand-specific antibodies. The invention also features receptor-specific antibodies which do not prevent ligand binding but prevent receptor activation. Receptor activation (i.e., signaling) may be determined by techniques described herein or otherwise known in the art. For example, receptor activation can be determined by detecting the phosphorylation (e.g., tyrosine or serine/threonine) of the receptor or its substrate by immunoprecipitation followed by western blot analysis (for example, as described supra). In specific embodiments, antibodies are provided that inhibit ligand activity or receptor activity by at least 95%, at least 90%, at least 85%, at least 80%, at least 75%, at least 75%, at least 70%, at least 50% of the activity in absence of the antibody.

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The invention also features receptor-specific antibodies which both prevent ligand binding and receptor activation as well as antibodies that recognize the receptor-ligand complex, and, preferably, do not specifically recognize the unbound receptor or the unbound ligand. Likewise, included in the invention are neutralizing antibodies which bind the ligand and prevent binding of the ligand to the receptor, as well as antibodies which bind the ligand, thereby preventing receptor activation, but do not prevent the ligand from binding the receptor. Further included in the invention are antibodies which activate the receptor. These antibodies may act as receptor agonists, i.e., potentiate or activate either all or a subset of the biological activities of the ligandmediated receptor activation, for example, by inducing dimerization of the receptor. antibodies may be specified as agonists, antagonists or inverse agonists for biological activities comprising the specific biological activities of the peptides of the invention disclosed herein. The above antibody agonists can be made using methods known in the art. See, e.g., PCT publication WO 96/40281; U.S. Patent No. 5,811,097; Deng et al., Blood 92(6):1981-1988 (1998); Chen et al., Cancer Res. 58(16):3668-3678 (1998); Harrop et al., J. Immunol. 161(4):1786-1794 (1998); Zhu et al., Cancer Res. 58(15):3209-3214 (1998); Yoon et al., J. Immunol. 160(7):3170-3179 (1998); Prat et al., J. Cell. Sci. 111(Pt2):237-247 (1998); Pitard et al., J. Immunol. Methods 205(2):177-190 (1997); Liautard et al., Cytokine 9(4):233-241 (1997); Carlson et al., J. Biol. Chem. 272(17):11295-11301 (1997); Taryman et al., Neuron 14(4):755-762 (1995); Muller et al., Structure 6(9):1153-1167 (1998); Bartunek et al., Cytokine 8(1):14-20 (1996) (which are all incorporated by reference herein in their entireties).

Antibodies of the present invention may be used, for example, to purify, detect, and target the polypeptides of the present invention, including both *in vitro* and *in vivo* diagnostic and therapeutic methods. For example, the antibodies have utility in immunoassays for qualitatively and quantitatively measuring levels of the polypeptides of the present invention in biological samples. See, e.g., Harlow et al., Antibodies: A Laboratory Manual, (Cold Spring Harbor Laboratory Press, 2nd ed. 1988); incorporated by reference herein in its entirety.

As discussed in more detail below, the antibodies of the present invention may be used either alone or in combination with other compositions. The antibodies may further be recombinantly fused to a heterologous polypeptide at the N- or C-terminus or chemically conjugated (including covalent and non-covalent conjugations) to polypeptides or other compositions. For example, antibodies of the present invention may be recombinantly fused or conjugated to molecules useful as labels in detection assays and effector molecules such as heterologous polypeptides, drugs, radionuclides, or toxins. See, e.g., PCT publications WO 92/08495; WO 91/14438; WO 89/12624; U.S. Patent No. 5,314,995; and EP 396,387; the disclosures of which are incorporated herein by reference in their entireties.

The antibodies of the invention include derivatives that are modified, i.e, by the covalent attachment of any type of molecule to the antibody such that covalent attachment does not prevent

the antibody from generating an anti-idiotypic response. For example, but not by way of limitation, the antibody derivatives include antibodies that have been modified, e.g., by glycosylation, acetylation, pegylation, phosphylation, amidation, derivatization by known protecting/blocking groups, proteolytic cleavage, linkage to a cellular ligand or other protein, etc. Any of numerous chemical modifications may be carried out by known techniques, including, but not limited to specific chemical cleavage, acetylation, formylation, metabolic synthesis of tunicamycin, etc. Additionally, the derivative may contain one or more non-classical amino acids.

The antibodies of the present invention may be generated by any suitable method known in the art. Polyclonal antibodies to an antigen-of- interest can be produced by various procedures well known in the art. For example, a polypeptide of the invention can be administered to various host animals including, but not limited to, rabbits, mice, rats, etc. to induce the production of sera containing polyclonal antibodies specific for the antigen. Various adjuvants may be used to increase the immunological response, depending on the host species, and include but are not limited to, Freund's (complete and incomplete), mineral gels such as aluminum hydroxide, surface active substances such as lysolecithin, pluronic polyols, polyanions, peptides, oil emulsions, keyhole limpet hemocyanins, dinitrophenol, and potentially useful human adjuvants such as BCG (bacille Calmette-Guerin) and corynebacterium parvum. Such adjuvants are also well known in the art.

Monoclonal antibodies can be prepared using a wide variety of techniques known in the art including the use of hybridoma, recombinant, and phage display technologies, or a combination thereof. For example, monoclonal antibodies can be produced using hybridoma techniques including those known in the art and taught, for example, in Harlow et al., Antibodies: A Laboratory Manual, (Cold Spring Harbor Laboratory Press, 2nd ed. 1988); Hammerling, et al., in: Monoclonal Antibodies and T-Cell Hybridomas 563-681 (Elsevier, N.Y., 1981) (said references incorporated by reference in their entireties). The term "monoclonal antibody" as used herein is not limited to antibodies produced through hybridoma technology. The term "monoclonal antibody" refers to an antibody that is derived from a single clone, including any eukaryotic, prokaryotic, or phage clone, and not the method by which it is produced.

Methods for producing and screening for specific antibodies using hybridoma technology are routine and well known in the art and are discussed in detail in the Examples. In a non-limiting example, mice can be immunized with a polypeptide of the invention or a cell expressing such peptide. Once an immune response is detected, e.g., antibodies specific for the antigen are detected in the mouse serum, the mouse spleen is harvested and splenocytes isolated. The splenocytes are then fused by well known techniques to any suitable myeloma cells, for example cells from cell line SP20 available from the ATCC. Hybridomas are selected and cloned by limited dilution. The hybridoma clones are then assayed by methods known in the art for cells that secrete antibodies capable of binding a polypeptide of the invention. Ascites fluid, which

generally contains high levels of antibodies, can be generated by immunizing mice with positive hybridoma clones.

Accordingly, the present invention provides methods of generating monoclonal antibodies as well as antibodies produced by the method comprising culturing a hybridoma cell secreting an antibody of the invention wherein, preferably, the hybridoma is generated by fusing splenocytes isolated from a mouse immunized with an antigen of the invention with myeloma cells and then screening the hybridomas resulting from the fusion for hybridoma clones that secrete an antibody able to bind a polypeptide of the invention.

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Another well known method for producing both polyclonal and monoclonal human B cell lines is transformation using Epstein Barr Virus (EBV). Protocols for generating EBV-transformed B cell lines are commonly known in the art, such as, for example, the protocol outlined in Chapter 7.22 of Current Protocols in Immunology, Coligan et al., Eds., 1994, John Wiley & Sons, NY, which is hereby incorporated in its entirety by reference. The source of B cells for transformation is commonly human peripheral blood, but B cells for transformation may also be derived from other sources including, but not limited to, lymph nodes, tonsil, spleen, tumor tissue, and infected tissues. Tissues are generally made into single cell suspensions prior to EBV transformation. Additionally, steps may be taken to either physically remove or inactivate T cells (e.g., by treatment with cyclosporin A) in B cell-containing samples, because T cells from individuals seropositive for anti-EBV antibodies can suppress B cell immortalization by EBV.

In general, the sample containing human B cells is innoculated with EBV, and cultured for 3-4 weeks. A typical source of EBV is the culture supernatant of the B95-8 cell line (ATCC #VR-1492). Physical signs of EBV transformation can generally be seen towards the end of the 3-4 week culture period. By phase-contrast microscopy, transformed cells may appear large, clear, hairy and tend to aggregate in tight clusters of cells. Initially, EBV lines are generally polyclonal. However, over prolonged periods of cell cultures, EBV lines may become monoclonal or polyclonal as a result of the selective outgrowth of particular B cell clones. Alternatively, polyclonal EBV transformed lines may be subcloned (e.g., by limiting dilution culture) or fused with a suitable fusion partner and plated at limiting dilution to obtain monoclonal B cell lines. Suitable fusion partners for EBV transformed cell lines include mouse myeloma cell lines (e.g., SP2/0, X63-Ag8.653), heteromyeloma cell lines (human x mouse; e.g, SPAM-8, SBC-H20, and CB-F7), and human cell lines (e.g., GM 1500, SKO-007, RPMI 8226, and KR-4). Thus, the present invention also provides a method of generating polyclonal or monoclonal human antibodies against polypeptides of the invention or fragments thereof, comprising EBV-transformation of human B cells.

Antibody fragments which recognize specific epitopes may be generated by known techniques. For example, Fab and F(ab')2 fragments of the invention may be produced by proteolytic cleavage of immunoglobulin molecules, using enzymes such as papain (to produce Fab

fragments) or pepsin (to produce F(ab')2 fragments). F(ab')2 fragments contain the variable region, the light chain constant region and the CH1 domain of the heavy chain.

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For example, the antibodies of the present invention can also be generated using various phage display methods known in the art. In phage display methods, functional antibody domains are displayed on the surface of phage particles which carry the polynucleotide sequences encoding them. In a particular embodiment, such phage can be utilized to display antigen binding domains expressed from a repertoire or combinatorial antibody library (e.g., human or murine). Phage expressing an antigen binding domain that binds the antigen of interest can be selected or identified with antigen, e.g., using labeled antigen or antigen bound or captured to a solid surface or bead. Phage used in these methods are typically filamentous phage including fd and M13 binding domains expressed from phage with Fab, Fv or disulfide stabilized Fv antibody domains recombinantly fused to either the phage gene III or gene VIII protein. Examples of phage display methods that can be used to make the antibodies of the present invention include those disclosed in Brinkman et al., J. Immunol. Methods 182:41-50 (1995); Ames et al., J. Immunol. Methods 184:177-186 (1995); Kettleborough et al., Eur. J. Immunol. 24:952-958 (1994); Persic et al., Gene 187 9-18 (1997); Burton et al., Advances in Immunology 57:191-280 (1994); PCT application No. PCT/GB91/01134; PCT publications WO 90/02809; WO 91/10737; WO 92/01047; WO 92/18619; WO 93/11236; WO 95/15982; WO 95/20401; and U.S. Patent Nos. 5,698,426; 5,223,409; 5,403,484; 5,580,717; 5,427,908; 5,750,753; 5,821,047; 5,571,698; 5,427,908; 5,516,637; 5,780,225; 5,658,727; 5,733,743 and 5,969,108; each of which is incorporated herein by reference in its entirety.

As described in the above references, after phage selection, the antibody coding regions from the phage can be isolated and used to generate whole antibodies, including human antibodies, or any other desired antigen binding fragment, and expressed in any desired host, including mammalian cells, insect cells, plant cells, yeast, and bacteria, e.g., as described in detail below. For example, techniques to recombinantly produce Fab, Fab' and F(ab')2 fragments can also be employed using methods known in the art such as those disclosed in PCT publication WO 92/22324; Mullinax et al., BioTechniques 12(6):864-869 (1992); and Sawai et al., AJRI 34:26-34 (1995); and Better et al., Science 240:1041-1043 (1988) (said references incorporated by reference in their entireties).

Examples of techniques which can be used to produce single-chain Fvs and antibodies include those described in U.S. Patents 4,946,778 and 5,258,498; Huston et al., Methods in Enzymology 203:46-88 (1991); Shu et al., PNAS 90:7995-7999 (1993); and Skerra et al., Science 240:1038-1040 (1988). For some uses, including *in vivo* use of antibodies in humans and in vitro detection assays, it may be preferable to use chimeric, humanized, or human antibodies. A chimeric antibody is a molecule in which different portions of the antibody are derived from different animal species, such as antibodies having a variable region derived from a murine

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monoclonal antibody and a human immunoglobulin constant region. Methods for producing chimeric antibodies are known in the art. See e.g., Morrison, Science 229:1202 (1985); Oi et al., BioTechniques 4:214 (1986); Gillies et al., (1989) J. Immunol. Methods 125:191-202; U.S. Patent Nos. 5,807,715; 4,816,567; and 4,816397, which are incorporated herein by reference in their entirety. Humanized antibodies are antibody molecules from non-human species antibody that binds the desired antigen having one or more complementarity determining regions (CDRs) from the non-human species and a framework regions from a human immunoglobulin molecule. Often, framework residues in the human framework regions will be substituted with the corresponding residue from the CDR donor antibody to alter, preferably improve, antigen binding. These framework substitutions are identified by methods well known in the art, e.g., by modeling of the interactions of the CDR and framework residues to identify framework residues important for antigen binding and sequence comparison to identify unusual framework residues at particular positions. (See, e.g., Queen et al., U.S. Patent No. 5,585,089; Riechmann et al., Nature 332:323 (1988), which are incorporated herein by reference in their entireties.) Antibodies can be humanized using a variety of techniques known in the art including, for example, CDR-grafting (EP 239,400; PCT publication WO 91/09967; U.S. Patent Nos. 5,225,539; 5,530,101; and 5,585,089), veneering or resurfacing (EP 592,106; EP 519,596; Padlan, Molecular Immunology 28(4/5):489-498 (1991); Studnicka et al., Protein Engineering 7(6):805-814 (1994); Roguska. et al., PNAS 91:969-973 (1994)), and chain shuffling (U.S. Patent No. 5,565,332).

Completely human antibodies are particularly desirable for therapeutic treatment of human patients. Human antibodies can be made by a variety of methods known in the art including phage display methods described above using antibody libraries derived from human immunoglobulin sequences. See also, U.S. Patent Nos. 4,444,887 and 4,716,111; and PCT publications WO 98/46645, WO 98/50433, WO 98/24893, WO 98/16654, WO 96/34096, WO 96/33735, and WO 91/10741; each of which is incorporated herein by reference in its entirety.

Human antibodies can also be produced using transgenic mice which are incapable of expressing functional endogenous immunoglobulins, but which can express human immunoglobulin genes. For example, the human heavy and light chain immunoglobulin gene complexes may be introduced randomly or by homologous recombination into mouse embryonic stem cells. Alternatively, the human variable region, constant region, and diversity region may be introduced into mouse embryonic stem cells in addition to the human heavy and light chain genes. The mouse heavy and light chain immunoglobulin genes may be rendered non-functional separately or simultaneously with the introduction of human immunoglobulin loci by homologous recombination. In particular, homozygous deletion of the JH region prevents endogenous antibody production. The modified embryonic stem cells are expanded and microinjected into blastocysts to produce chimeric mice. The chimeric mice are then bred to produce homozygous offspring which express human antibodies. The transgenic mice are immunized in the normal fashion with a

selected antigen, e.g., all or a portion of a polypeptide of the invention. Monoclonal antibodies directed against the antigen can be obtained from the immunized, transgenic mice using conventional hybridoma technology. The human immunoglobulin transgenes harbored by the transgenic mice rearrange during B cell differentiation, and subsequently undergo class switching and somatic mutation. Thus, using such a technique, it is possible to produce therapeutically useful IgG, IgA, IgM and IgE antibodies. For an overview of this technology for producing human antibodies, see Lonberg and Huszar, Int. Rev. Immunol. 13:65-93 (1995). For a detailed discussion of this technology for producing human antibodies and human monoclonal antibodies and protocols for producing such antibodies, see, e.g., PCT publications WO 98/24893; WO 92/01047; WO 96/34096; WO 96/33735; European Patent No. 0 598 877; U.S. Patent Nos. 5,413,923; 5,625,126; 5,633,425; 5,569,825; 5,661,016; 5,545,806; 5,814,318; 5,885,793; 5,916,771; 5,939,598; 6,075,181; and 6,114,598, which are incorporated by reference herein in their entirety. In addition, companies such as Abgenix, Inc. (Freemont, CA) and Genpharm (San Jose, CA) can be engaged to provide human antibodies directed against a selected antigen using technology similar to that described above.

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Completely human antibodies which recognize a selected epitope can be generated using a technique referred to as "guided selection." In this approach a selected non-human monoclonal antibody, e.g., a mouse antibody, is used to guide the selection of a completely human antibody recognizing the same epitope. (Jespers et al., Bio/technology 12:899-903 (1988)).

Further, antibodies to the polypeptides of the invention can, in turn, be utilized to generate anti-idiotype antibodies that "mimic" polypeptides of the invention using techniques well known to those skilled in the art. (See, e.g., Greenspan & Bona, FASEB J. 7(5):437-444; (1989) and Nissinoff, J. Immunol. 147(8):2429-2438 (1991)). For example, antibodies which bind to and competitively inhibit polypeptide multimerization and/or binding of a polypeptide of the invention to a ligand can be used to generate anti-idiotypes that "mimic" the polypeptide multimerization and/or binding domain and, as a consequence, bind to and neutralize polypeptide and/or its ligand. Such neutralizing anti-idiotypes or Fab fragments of such anti-idiotypes can be used in therapeutic regimens to neutralize polypeptide ligand(s)/receptor(s). For example, such anti-idiotypic antibodies can be used to bind a polypeptide of the invention and/or to bind its ligand(s)/receptor(s), and thereby block its biological activity. Alternatively, antibodies which bind to and enhance polypeptide multimerization and/or binding, and/or receptor/ligand multimerization, binding and/or signaling can be used to generate anti-idiotypes that function as agonists of a polypeptide of the invention and/or its ligand/receptor. Such agonistic anti-idiotypes or Fab fragments of such anti-idiotypes can be used in therapeutic regimens as agonists of the polypeptides of the invention or its ligand(s)/receptor(s). For example, such anti-idiotypic antibodies can be used to bind a polypeptide of the invention and/or to bind its ligand(s)/receptor(s), and thereby promote or enhance its biological activity.

Intrabodies of the invention can be produced using methods known in the art, such as those disclosed and reviewed in Chen et al., Hum. Gene Ther. 5:595-601 (1994); Marasco, W.A., Gene Ther. 4:11-15 (1997); Rondon and Marasco, Annu. Rev. Microbiol. 51:257-283 (1997); Proba et al., J. Mol. Biol. 275:245-253 (1998); Cohen et al., Oncogene 17:2445-2456 (1998); Ohage and Steipe, J. Mol. Biol. 291:1119-1128 (1999); Ohage et al., J. Mol. Biol. 291:1129-1134 (1999); Wirtz and Steipe, Protein Sci. 8:2245-2250 (1999); Zhu et al., J. Immunol. Methods 231:207-222 (1999); and references cited therein.

### Polynucleotides Encoding Antibodies

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The invention further provides polynucleotides comprising a nucleotide sequence encoding an antibody of the invention and fragments thereof. The invention also encompasses polynucleotides that hybridize under stringent or alternatively, under lower stringency hybridization conditions, e.g., as defined *supra*, to polynucleotides that encode an antibody, preferably, that specifically binds to a polypeptide of the invention, preferably, an antibody that binds to a polypeptide having the amino acid sequence of SEQ ID NO:Y, to a polypeptide encoded by a portion of SEQ ID NO:X as defined in columns 8 and 9 of Table 2, and/or to a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z.

The polynucleotides may be obtained, and the nucleotide sequence of the polynucleotides determined, by any method known in the art. For example, if the nucleotide sequence of the antibody is known, a polynucleotide encoding the antibody may be assembled from chemically synthesized oligonucleotides (e.g., as described in Kutmeier et al., BioTechniques 17:242 (1994)), which, briefly, involves the synthesis of overlapping oligonucleotides containing portions of the sequence encoding the antibody, annealing and ligating of those oligonucleotides, and then amplification of the ligated oligonucleotides by PCR.

Alternatively, a polynucleotide encoding an antibody may be generated from nucleic acid from a suitable source. If a clone containing a nucleic acid encoding a particular antibody is not available, but the sequence of the antibody molecule is known, a nucleic acid encoding the immunoglobulin may be chemically synthesized or obtained from a suitable source (e.g., an antibody cDNA library, or a cDNA library generated from, or nucleic acid, preferably poly A+RNA, isolated from, any tissue or cells expressing the antibody, such as hybridoma cells selected to express an antibody of the invention) by PCR amplification using synthetic primers hybridizable to the 3' and 5' ends of the sequence or by cloning using an oligonucleotide probe specific for the particular gene sequence to identify, e.g., a cDNA clone from a cDNA library that encodes the antibody. Amplified nucleic acids generated by PCR may then be cloned into replicable cloning vectors using any method well known in the art.

Once the nucleotide sequence and corresponding amino acid sequence of the antibody is determined, the nucleotide sequence of the antibody may be manipulated using methods well

known in the art for the manipulation of nucleotide sequences, e.g., recombinant DNA techniques, site directed mutagenesis, PCR, etc. (see, for example, the techniques described in Sambrook et al., 1990, Molecular Cloning, A Laboratory Manual, 2d Ed., Cold Spring Harbor Laboratory, Cold Spring Harbor, NY and Ausubel et al., eds., 1998, Current Protocols in Molecular Biology, John Wiley & Sons, NY, which are both incorporated by reference herein in their entireties ), to generate antibodies having a different amino acid sequence, for example to create amino acid substitutions, deletions, and/or insertions.

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In a specific embodiment, the amino acid sequence of the heavy and/or light chain variable domains may be inspected to identify the sequences of the complementarity determining regions (CDRs) by methods that are well know in the art, e.g., by comparison to known amino acid sequences of other heavy and light chain variable regions to determine the regions of sequence hypervariability. Using routine recombinant DNA techniques, one or more of the CDRs may be inserted within framework regions, e.g., into human framework regions to humanize a non-human antibody, as described supra. The framework regions may be naturally occurring or consensus framework regions, and preferably human framework regions (see, e.g., Chothia et al., J. Mol. Biol. 278: 457-479 (1998) for a listing of human framework regions). Preferably, the polynucleotide generated by the combination of the framework regions and CDRs encodes an antibody that specifically binds a polypeptide of the invention. Preferably, as discussed supra, one or more amino acid substitutions may be made within the framework regions, and, preferably, the amino acid substitutions improve binding of the antibody to its antigen. Additionally, such methods may be used to make amino acid substitutions or deletions of one or more variable region cysteine residues participating in an intrachain disulfide bond to generate antibody molecules lacking one or more intrachain disulfide bonds. Other alterations to the polynucleotide are encompassed by the present invention and within the skill of the art.

In addition, techniques developed for the production of "chimeric antibodies" (Morrison et al., Proc. Natl. Acad. Sci. 81:851-855 (1984); Neuberger et al., Nature 312:604-608 (1984); Takeda et al., Nature 314:452-454 (1985)) by splicing genes from a mouse antibody molecule of appropriate antigen specificity together with genes from a human antibody molecule of appropriate biological activity can be used. As described *supra*, a chimeric antibody is a molecule in which different portions are derived from different animal species, such as those having a variable region derived from a murine mAb and a human immunoglobulin constant region, e.g., humanized antibodies.

Alternatively, techniques described for the production of single chain antibodies (U.S. Patent No. 4,946,778; Bird, Science 242:423-42 (1988); Huston et al., Proc. Natl. Acad. Sci. USA 85:5879-5883 (1988); and Ward et al., Nature 334:544-54 (1989)) can be adapted to produce single chain antibodies. Single chain antibodies are formed by linking the heavy and light chain fragments of the Fv region via an amino acid bridge, resulting in a single chain polypeptide.

Techniques for the assembly of functional Fv fragments in E. coli may also be used (Skerra et al., Science 242:1038-1041 (1988)).

# Methods of Producing Antibodies

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The antibodies of the invention can be produced by any method known in the art for the synthesis of antibodies, in particular, by chemical synthesis or preferably, by recombinant expression techniques. Methods of producing antibodies include, but are not limited to, hybridoma technology, EBV transformation, and other methods discussed herein as well as through the use recombinant DNA technology, as discussed below.

Recombinant expression of an antibody of the invention, or fragment, derivative or analog thereof, (e.g., a heavy or light chain of an antibody of the invention or a single chain antibody of the invention), requires construction of an expression vector containing a polynucleotide that encodes the antibody. Once a polynucleotide encoding an antibody molecule or a heavy or light chain of an antibody, or portion thereof (preferably containing the heavy or light chain variable domain), of the invention has been obtained, the vector for the production of the antibody molecule may be produced by recombinant DNA technology using techniques well known in the art. Thus, methods for preparing a protein by expressing a polynucleotide containing an antibody encoding nucleotide sequence are described herein. Methods which are well known to those skilled in the art can be used to construct expression vectors containing antibody coding sequences and appropriate transcriptional and translational control signals. These methods include, for example, in vitro recombinant DNA techniques, synthetic techniques, and in vivo genetic recombination. The invention, thus, provides replicable vectors comprising a nucleotide sequence encoding an antibody molecule of the invention, or a heavy or light chain thereof, or a heavy or light chain variable domain, operably linked to a promoter. Such vectors may include the nucleotide sequence encoding the constant region of the antibody molecule (see, e.g., PCT Publication WO 86/05807; PCT Publication WO 89/01036; and U.S. Patent No. 5,122,464) and the variable domain of the antibody may be cloned into such a vector for expression of the entire heavy or light chain.

The expression vector is transferred to a host cell by conventional techniques and the transfected cells are then cultured by conventional techniques to produce an antibody of the invention. Thus, the invention includes host cells containing a polynucleotide encoding an antibody of the invention, or a heavy or light chain thereof, or a single chain antibody of the invention, operably linked to a heterologous promoter. In preferred embodiments for the expression of double-chained antibodies, vectors encoding both the heavy and light chains may be co-expressed in the host cell for expression of the entire immunoglobulin molecule, as detailed below.

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A variety of host-expression vector systems may be utilized to express the antibody molecules of the invention. Such host-expression systems represent vehicles by which the coding sequences of interest may be produced and subsequently purified, but also represent cells which may, when transformed or transfected with the appropriate nucleotide coding sequences, express an antibody molecule of the invention in situ. These include but are not limited to microorganisms such as bacteria (e.g., E. coli, B. subtilis) transformed with recombinant bacteriophage DNA, plasmid DNA or cosmid DNA expression vectors containing antibody coding sequences; yeast (e.g., Saccharomyces, Pichia) transformed with recombinant yeast expression vectors containing antibody coding sequences; insect cell systems infected with recombinant virus expression vectors (e.g., baculovirus) containing antibody coding sequences; plant cell systems infected with recombinant virus expression vectors (e.g., cauliflower mosaic virus, CaMV; tobacco mosaic virus, TMV) or transformed with recombinant plasmid expression vectors (e.g., Ti plasmid) containing antibody coding sequences; or mammalian cell systems (e.g., COS, CHO, BHK, 293, 3T3 cells) harboring recombinant expression constructs containing promoters derived from the genome of mammalian cells (e.g., metallothionein promoter) or from mammalian viruses (e.g., the adenovirus late promoter; the vaccinia virus 7.5K promoter). Preferably, bacterial cells such as Escherichia coli, and more preferably, eukaryotic cells, especially for the expression of whole recombinant antibody molecule, are used for the expression of a recombinant antibody molecule. For example, mammalian cells such as Chinese hamster ovary cells (CHO), in conjunction with a vector such as the major intermediate early gene promoter element from human cytomegalovirus is an effective expression system for antibodies (Foecking et al., Gene 45:101 (1986); Cockett et al., Bio/Technology 8:2 (1990)).

In bacterial systems, a number of expression vectors may be advantageously selected depending upon the use intended for the antibody molecule being expressed. For example, when a large quantity of such a protein is to be produced, for the generation of pharmaceutical compositions of an antibody molecule, vectors which direct the expression of high levels of fusion protein products that are readily purified may be desirable. Such vectors include, but are not limited, to the E. coli expression vector pUR278 (Ruther et al., EMBO J. 2:1791 (1983)), in which the antibody coding sequence may be ligated individually into the vector in frame with the lac Z coding region so that a fusion protein is produced; pIN vectors (Inouye & Inouye, Nucleic Acids Res. 13:3101-3109 (1985); Van Heeke & Schuster, J. Biol. Chem. 24:5503-5509 (1989)); and the like. pGEX vectors may also be used to express foreign polypeptides as fusion proteins with glutathione S-transferase (GST). In general, such fusion proteins are soluble and can easily be purified from lysed cells by adsorption and binding to matrix glutathione-agarose beads followed by elution in the presence of free glutathione. The pGEX vectors are designed to include thrombin or factor Xa protease cleavage sites so that the cloned target gene product can be released from the GST moiety.

In an insect system, Autographa californica nuclear polyhedrosis virus (AcNPV) is used as a vector to express foreign genes. The virus grows in *Spodoptera frugiperda* cells. The antibody coding sequence may be cloned individually into non-essential regions (for example the polyhedrin gene) of the virus and placed under control of an AcNPV promoter (for example the polyhedrin promoter).

In mammalian host cells, a number of viral-based expression systems may be utilized. In cases where an adenovirus is used as an expression vector, the antibody coding sequence of interest may be ligated to an adenovirus transcription/translation control complex, e.g., the late promoter and tripartite leader sequence. This chimeric gene may then be inserted in the adenovirus genome by in vitro or *in vivo* recombination. Insertion in a non- essential region of the viral genome (e.g., region E1 or E3) will result in a recombinant virus that is viable and capable of expressing the antibody molecule in infected hosts. (e.g., see Logan & Shenk, Proc. Natl. Acad. Sci. USA 81:355-359 (1984)). Specific initiation signals may also be required for efficient translation of inserted antibody coding sequences. These signals include the ATG initiation codon and adjacent sequences. Furthermore, the initiation codon must be in phase with the reading frame of the desired coding sequence to ensure translation of the entire insert. These exogenous translational control signals and initiation codons can be of a variety of origins, both natural and synthetic. The efficiency of expression may be enhanced by the inclusion of appropriate transcription enhancer elements, transcription terminators, etc. (see Bittner et al., Methods in Enzymol. 153:51-544 (1987)).

In addition, a host cell strain may be chosen which modulates the expression of the inserted sequences, or modifies and processes the gene product in the specific fashion desired. Such modifications (e.g., glycosylation) and processing (e.g., cleavage) of protein products may be important for the function of the protein. Different host cells have characteristic and specific mechanisms for the post-translational processing and modification of proteins and gene products. Appropriate cell lines or host systems can be chosen to ensure the correct modification and processing of the foreign protein expressed. To this end, eukaryotic host cells which possess the cellular machinery for proper processing of the primary transcript, glycosylation, and phosphorylation of the gene product may be used. Such mammalian host cells include but are not limited to CHO, VERY, BHK, Hela, COS, MDCK, 293, 3T3, WI38, and in particular, breast cancer cell lines such as, for example, BT483, Hs578T, HTB2, BT20 and T47D, and normal mammary gland cell line such as, for example, CRL7030 and Hs578Bst.

For long-term, high-yield production of recombinant proteins, stable expression is preferred. For example, cell lines which stably express the antibody molecule may be engineered. Rather than using expression vectors which contain viral origins of replication, host cells can be transformed with DNA controlled by appropriate expression control elements (e.g., promoter, enhancer, sequences, transcription terminators, polyadenylation sites, etc.), and a selectable

marker. Following the introduction of the foreign DNA, engineered cells may be allowed to grow for 1-2 days in an enriched media, and then are switched to a selective media. The selectable marker in the recombinant plasmid confers resistance to the selection and allows cells to stably integrate the plasmid into their chromosomes and grow to form foci which in turn can be cloned and expanded into cell lines. This method may advantageously be used to engineer cell lines which express the antibody molecule. Such engineered cell lines may be particularly useful in screening and evaluation of compounds that interact directly or indirectly with the antibody molecule.

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A number of selection systems may be used, including but not limited to the herpes simplex virus thymidine kinase (Wigler et al., Cell 11:223 (1977)), hypoxanthine-guanine phosphoribosyltransferase (Szybalska & Szybalski, Proc. Natl. Acad. Sci. USA 48:202 (1992)), and adenine phosphoribosyltransferase (Lowy et al., Cell 22:817 (1980)) genes can be employed in tk-, hgprt- or aprt- cells, respectively. Also, antimetabolite resistance can be used as the basis of selection for the following genes: dhfr, which confers resistance to methotrexate (Wigler et al., Natl. Acad. Sci. USA 77:357 (1980); O'Hare et al., Proc. Natl. Acad. Sci. USA 78:1527 (1981)); gpt, which confers resistance to mycophenolic acid (Mulligan & Berg, Proc. Natl. Acad. Sci. USA 78:2072 (1981)); neo, which confers resistance to the aminoglycoside G-418 Clinical Pharmacy 12:488-505; Wu and Wu, Biotherapy 3:87-95 (1991); Tolstoshev, Ann. Rev. Pharmacol. Toxicol. 32:573-596 (1993); Mulligan, Science 260:926-932 (1993); and Morgan and Anderson, Ann. Rev. Biochem. 62:191-217 (1993); May, 1993, TIB TECH 11(5):155-215 (1993)); and hygro, which confers resistance to hygromycin (Santerre et al., Gene 30:147 (1984)). Methods commonly known in the art of recombinant DNA technology may be routinely applied to select the desired recombinant clone, and such methods are described, for example, in Ausubel et al. (eds.), Current Protocols in Molecular Biology, John Wiley & Sons, NY (1993); Kriegler, Gene Transfer and Expression, A Laboratory Manual, Stockton Press, NY (1990); and in Chapters 12 and 13, Dracopoli et al. (eds), Current Protocols in Human Genetics, John Wiley & Sons, NY (1994); Colberre-Garapin et al., J. Mol. Biol. 150:1 (1981), which are incorporated by reference herein in their entireties.

The expression levels of an antibody molecule can be increased by vector amplification (for a review, see Bebbington and Hentschel, The use of vectors based on gene amplification for the expression of cloned genes in mammalian cells in DNA cloning, Vol.3. (Academic Press, New York, 1987)). When a marker in the vector system expressing antibody is amplifiable, increase in the level of inhibitor present in culture of host cell will increase the number of copies of the marker gene. Since the amplified region is associated with the antibody gene, production of the antibody will also increase (Crouse et al., Mol. Cell. Biol, 3:257 (1983)).

Vectors which use glutamine synthase (GS) or DHFR as the selectable markers can be amplified in the presence of the drugs methionine sulphoximine or methotrexate, respectively. An

advantage of glutamine synthase based vectors are the availabilty of cell lines (e.g., the murine myeloma cell line, NS0) which are glutamine synthase negative. Glutamine synthase expression systems can also function in glutamine synthase expressing cells (e.g. Chinese Hamster Ovary (CHO) cells) by providing additional inhibitor to prevent the functioning of the endogenous gene. A glutamine synthase expression system and components thereof are detailed in PCT publications: WO87/04462; WO86/05807; WO89/01036; WO89/10404; and WO91/06657 which are incorporated in their entireties by reference herein. Additionally, glutamine synthase expression vectors that may be used according to the present invention are commercially available from suplliers, including, for example Lonza Biologics, Inc. (Portsmouth, NH). Expression and production of monoclonal antibodies using a GS expression system in murine myeloma cells is described in Bebbington et al., Bio/technology 10:169(1992) and in Biblia and Robinson Biotechnol. Prog. 11:1 (1995) which are incorporated in their entirities by reference herein.

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The host cell may be co-transfected with two expression vectors of the invention, the first vector encoding a heavy chain derived polypeptide and the second vector encoding a light chain derived polypeptide. The two vectors may contain identical selectable markers which enable equal expression of heavy and light chain polypeptides. Alternatively, a single vector may be used which encodes, and is capable of expressing, both heavy and light chain polypeptides. In such situations, the light chain should be placed before the heavy chain to avoid an excess of toxic free heavy chain (Proudfoot, Nature 322:52 (1986); Kohler, Proc. Natl. Acad. Sci. USA 77:2197 (1980)). The coding sequences for the heavy and light chains may comprise cDNA or genomic DNA.

Once an antibody molecule of the invention has been produced by an animal, chemically synthesized, or recombinantly expressed, it may be purified by any method known in the art for purification of an immunoglobulin molecule, for example, by chromatography (e.g., ion exchange, affinity, particularly by affinity for the specific antigen after Protein A, and sizing column chromatography), centrifugation, differential solubility, or by any other standard technique for the purification of proteins. In addition, the antibodies of the present invention or fragments thereof can be fused to heterologous polypeptide sequences described herein or otherwise known in the art, to facilitate purification.

The present invention encompasses antibodies recombinantly fused or chemically conjugated (including both covalently and non-covalently conjugations) to a polypeptide (or portion thereof, preferably at least 10, 20, 30, 40, 50, 60, 70, 80, 90 or 100 amino acids of the polypeptide) of the present invention to generate fusion proteins. The fusion does not necessarily need to be direct, but may occur through linker sequences. The antibodies may be specific for antigens other than polypeptides (or portion thereof, preferably at least 10, 20, 30, 40, 50, 60, 70, 80, 90 or 100 amino acids of the polypeptide) of the present invention. For example, antibodies may be used to target the polypeptides of the present invention to particular cell types, either in

vitro or *in vivo*, by fusing or conjugating the polypeptides of the present invention to antibodies specific for particular cell surface receptors. Antibodies fused or conjugated to the polypeptides of the present invention may also be used in in vitro immunoassays and purification methods using methods known in the art. See e.g., Harbor et al., *supra*, and PCT publication WO 93/21232; EP 439,095; Naramura et al., Immunol. Lett. 39:91-99 (1994); U.S. Patent 5,474,981; Gillies et al., PNAS 89:1428-1432 (1992); Fell et al., J. Immunol. 146:2446-2452 (1991), which are incorporated by reference in their entireties.

The present invention further includes compositions comprising the polypeptides of the present invention fused or conjugated to antibody domains other than the variable regions. For example, the polypeptides of the present invention may be fused or conjugated to an antibody Fc region, or portion thereof. The antibody portion fused to a polypeptide of the present invention may comprise the constant region, hinge region, CH1 domain, CH2 domain, and CH3 domain or any combination of whole domains or portions thereof. The polypeptides may also be fused or conjugated to the above antibody portions to form multimers. For example, Fc portions fused to the polypeptides of the present invention can form dimers through disulfide bonding between the Fc portions. Higher multimeric forms can be made by fusing the polypeptides to portions of IgA and IgM. Methods for fusing or conjugating the polypeptides of the present invention to antibody portions are known in the art. See, e.g., U.S. Patent Nos. 5,336,603; 5,622,929; 5,359,046; 5,349,053; 5,447,851; 5,112,946; EP 307,434; EP 367,166; PCT publications WO 96/04388; WO 91/06570; Ashkenazi et al., Proc. Natl. Acad. Sci. USA 88:10535-10539 (1991); Zheng et al., J. Immunol. 154:5590-5600 (1995); and Vil et al., Proc. Natl. Acad. Sci. USA 89:11337- 11341 (1992) (said references incorporated by reference in their entireties).

As discussed, *supra*, the polypeptides corresponding to a polypeptide, polypeptide fragment, or a variant of SEQ ID NO:Y may be fused or conjugated to the above antibody portions to increase the *in vivo* half life of the polypeptides or for use in immunoassays using methods known in the art. Further, the polypeptides corresponding to SEQ ID NO:Y may be fused or conjugated to the above antibody portions to facilitate purification. One reported example describes chimeric proteins consisting of the first two domains of the human CD4-polypeptide and various domains of the constant regions of the heavy or light chains of mammalian immunoglobulins. See EP 394,827; and Traunecker et al., Nature 331:84-86 (1988). The polypeptides of the present invention fused or conjugated to an antibody having disulfide-linked dimeric structures (due to the IgG) may also be more efficient in binding and neutralizing other molecules, than the monomeric secreted protein or protein fragment alone. See, for example, Fountoulakis et al., J. Biochem. 270:3958-3964 (1995). In many cases, the Fc part in a fusion protein is beneficial in therapy and diagnosis, and thus can result in, for example, improved pharmacokinetic properties. See, for example, EP A 232,262. Alternatively, deleting the Fc part after the fusion protein has been expressed, detected, and purified, would be desired. For example,

the Fc portion may hinder therapy and diagnosis if the fusion protein is used as an antigen for immunizations. In drug discovery, for example, human proteins, such as hIL-5, have been fused with Fc portions for the purpose of high-throughput screening assays to identify antagonists of hIL-5. (See, Bennett et al., J. Molecular Recognition 8:52-58 (1995); Johanson et al., J. Biol. Chem. 270:9459-9471 (1995)).

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Moreover, the antibodies or fragments thereof of the present invention can be fused to marker sequences, such as a peptide to facilitate purification. In preferred embodiments, the marker amino acid sequence is a hexa-histidine peptide, such as the tag provided in a pQE vector (QIAGEN, Inc., 9259 Eton Avenue, Chatsworth, CA, 91311), among others, many of which are commercially available. As described in Gentz et al., Proc. Natl. Acad. Sci. USA 86:821-824 (1989), for instance, hexa-histidine provides for convenient purification of the fusion protein. Other peptide tags useful for purification include, but are not limited to, the "HA" tag, which corresponds to an epitope derived from the influenza hemagglutinin protein (Wilson et al., Cell 37:767 (1984)) and the "flag" tag.

The present invention further encompasses antibodies or fragments thereof conjugated to a diagnostic or therapeutic agent. The antibodies can be used diagnostically to, for example, monitor the development or progression of a tumor as part of a clinical testing procedure to, e.g., determine the efficacy of a given treatment regimen. Detection can be facilitated by coupling the antibody to a detectable substance. Examples of detectable substances include various enzymes, prosthetic groups, fluorescent materials, luminescent materials, bioluminescent materials, radioactive materials, positron emitting metals using various positron emission tomographies, and nonradioactive paramagnetic metal ions. The detectable substance may be coupled or conjugated either directly to the antibody (or fragment thereof) or indirectly, through an intermediate (such as, for example, a linker known in the art) using techniques known in the art. See, for example, U.S. Patent No. 4,741,900 for metal ions which can be conjugated to antibodies for use as diagnostics according to the present invention. Examples of suitable enzymes include horseradish peroxidase, alkaline phosphatase, beta-galactosidase, or acetylcholinesterase; examples of suitable prosthetic group complexes include streptavidin/biotin and avidin/biotin; examples of suitable fluorescent materials include umbelliferone, fluorescein, fluorescein isothiocyanate, rhodamine, dichlorotriazinylamine fluorescein, dansyl chloride or phycoerythrin; an example of a luminescent material includes luminol; examples of bioluminescent materials include luciferase, luciferin, and aequorin; and examples of suitable radioactive material include 125I, 131I, 111In or 99Tc.

Further, an antibody or fragment thereof may be conjugated to a therapeutic moiety such as a cytotoxin, e.g., a cytostatic or cytocidal agent, a therapeutic agent or a radioactive metal ion, e.g., alpha-emitters such as, for example, 213Bi. A cytotoxin or cytotoxic agent includes any agent that is detrimental to cells. Examples include paclitaxol, cytochalasin B, gramicidin D, ethidium bromide, emetine, mitomycin, etoposide, tenoposide, vincristine, vinblastine, colchicin,

doxorubicin, daunorubicin, dihydroxy anthracin dione, mitoxantrone, mithramycin, actinomycin D, 1-dehydrotestosterone, glucocorticoids, procaine, tetracaine, lidocaine, propranolol, and puromycin and analogs or homologs thereof. Therapeutic agents include, but are not limited to, antimetabolites (e.g., methotrexate, 6-mercaptopurine, 6-thioguanine, cytarabine, 5-fluorouracil decarbazine), alkylating agents (e.g., mechlorethamine, thioepa chlorambucil, melphalan, carmustine (BSNU) and lomustine (CCNU), cyclothosphamide, busulfan, dibromomannitol, streptozotocin, mitomycin C, and cis- dichlorodiamine platinum (II) (DDP) cisplatin), anthracyclines (e.g., daunorubicin (formerly daunomycin) and doxorubicin), antibiotics (e.g., dactinomycin (formerly actinomycin), bleomycin, mithramycin, and anthramycin (AMC)), and anti-mitotic agents (e.g., vincristine and vinblastine).

The conjugates of the invention can be used for modifying a given biological response, the therapeutic agent or drug moiety is not to be construed as limited to classical chemical therapeutic agents. For example, the drug moiety may be a protein or polypeptide possessing a desired biological activity. Such proteins may include, for example, a toxin such as abrin, ricin A, pseudomonas exotoxin, or diphtheria toxin; a protein such as tumor necrosis factor, a-interferon, β-interferon, nerve growth factor, platelet derived growth factor, tissue plasminogen activator, an apoptotic agent, e.g., TNF-alpha, TNF-beta, AIM I (See, International Publication No. WO 97/33899), AIM II (See, International Publication No. WO 97/34911), Fas Ligand (Takahashi et al., Int. Immunol., 6:1567-1574 (1994)), VEGI (See, International Publication No. WO 99/23105), a thrombotic agent or an anti- angiogenic agent, e.g., angiostatin or endostatin; or, biological response modifiers such as, for example, lymphokines, interleukin-1 ("IL-1"), interleukin-2 ("IL-2"), interleukin-6 ("IL-6"), granulocyte macrophage colony stimulating factor ("GM-CSF"), granulocyte colony stimulating factor ("G-CSF"), or other growth factors.

Antibodies may also be attached to solid supports, which are particularly useful for immunoassays or purification of the target antigen. Such solid supports include, but are not limited to, glass, cellulose, polyacrylamide, nylon, polystyrene, polyvinyl chloride or polypropylene.

Techniques for conjugating such therapeutic moiety to antibodies are well known. See, for example, Arnon et al., "Monoclonal Antibodies For Immunotargeting Of Drugs In Cancer Therapy", in Monoclonal Antibodies And Cancer Therapy, Reisfeld et al. (eds.), pp. 243-56 (Alan R. Liss, Inc. 1985); Hellstrom et al., "Antibodies For Drug Delivery", in Controlled Drug Delivery (2nd Ed.), Robinson et al. (eds.), pp. 623-53 (Marcel Dekker, Inc. 1987); Thorpe, "Antibody Carriers Of Cytotoxic Agents In Cancer Therapy: A Review", in Monoclonal Antibodies '84: Biological And Clinical Applications, Pinchera et al. (eds.), pp. 475-506 (1985); "Analysis, Results, And Future Prospective Of The Therapeutic Use Of Radiolabeled Antibody In Cancer Therapy", in Monoclonal Antibodies For Cancer Detection And Therapy, Baldwin et al.

(eds.), pp. 303-16 (Academic Press 1985), and Thorpe et al., "The Preparation And Cytotoxic Properties Of Antibody-Toxin Conjugates", Immunol. Rev. 62:119-58 (1982).

Alternatively, an antibody can be conjugated to a second antibody to form an antibody heteroconjugate as described by Segal in U.S. Patent No. 4,676,980, which is incorporated herein by reference in its entirety.

An antibody, with or without a therapeutic moiety conjugated to it, administered alone or in combination with cytotoxic factor(s) and/or cytokine(s) can be used as a therapeutic.

## **Immunophenotyping**

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The antibodies of the invention may be utilized for immunophenotyping of cell lines and biological samples. Translation products of the gene of the present invention may be useful as cell-specific markers, or more specifically as cellular markers that are differentially expressed at various stages of differentiation and/or maturation of particular cell types. Monoclonal antibodies directed against a specific epitope, or combination of epitopes, will allow for the screening of cellular populations expressing the marker. Various techniques can be utilized using monoclonal antibodies to screen for cellular populations expressing the marker(s), and include magnetic separation using antibody-coated magnetic beads, "panning" with antibody attached to a solid matrix (i.e., plate), and flow cytometry (See, e.g., U.S. Patent 5,985,660; and Morrison et al., Cell, 96:737-49 (1999)).

These techniques allow for the screening of particular populations of cells, such as might be found with hematological malignancies (i.e. minimal residual disease (MRD) in acute leukemic patients) and "non-self" cells in transplantations to prevent Graft-versus-Host Disease (GVHD). Alternatively, these techniques allow for the screening of hematopoietic stem and progenitor cells capable of undergoing proliferation and/or differentiation, as might be found in human umbilical cord blood.

### Assays For Antibody Binding

The antibodies of the invention may be assayed for immunospecific binding by any method known in the art. The immunoassays which can be used include but are not limited to competitive and non-competitive assay systems using techniques such as western blots, radioimmunoassays, ELISA (enzyme linked immunosorbent assay), "sandwich" immunoassays, immunoprecipitation assays, precipitin reactions, gel diffusion precipitin reactions, immunodiffusion assays, agglutination assays, complement-fixation assays, immunoradiometric assays, fluorescent immunoassays, and protein A immunoassays, to name but a few. Such assays are routine and well known in the art (see, e.g., Ausubel et al, eds, 1994, Current Protocols in Molecular Biology, Vol. 1, John Wiley & Sons, Inc., New York, which is incorporated by

reference herein in its entirety). Exemplary immunoassays are described briefly below (but are not intended by way of limitation).

Immunoprecipitation protocols generally comprise lysing a population of cells in a lysis buffer such as RIPA buffer (1% NP-40 or Triton X- 100, 1% sodium deoxycholate, 0.1% SDS, 0.15 M NaCl, 0.01 M sodium phosphate at pH 7.2, 1% Trasylol) supplemented with protein phosphatase and/or protease inhibitors (e.g., EDTA, PMSF, aprotinin, sodium vanadate), adding the antibody of interest to the cell lysate, incubating for a period of time (e.g., 1-4 hours) at 4° C, adding protein A and/or protein G sepharose beads to the cell lysate, incubating for about an hour or more at 4° C, washing the beads in lysis buffer and resuspending the beads in SDS/sample buffer. The ability of the antibody of interest to immunoprecipitate a particular antigen can be assessed by, e.g., western blot analysis. One of skill in the art would be knowledgeable as to the parameters that can be modified to increase the binding of the antibody to an antigen and decrease the background (e.g., pre-clearing the cell lysate with sepharose beads). For further discussion regarding immunoprecipitation protocols see, e.g., Ausubel et al., eds., (1994), Current Protocols in Molecular Biology, Vol. 1, John Wiley & Sons, Inc., New York, section 10.16.1.

Western blot analysis generally comprises preparing protein samples, electrophoresis of the protein samples in a polyacrylamide gel (e.g., 8%- 20% SDS-PAGE depending on the molecular weight of the antigen), transferring the protein sample from the polyacrylamide gel to a membrane such as nitrocellulose, PVDF or nylon, blocking the membrane in blocking solution (e.g., PBS with 3% BSA or non-fat milk), washing the membrane in washing buffer (e.g., PBS-Tween 20), blocking the membrane with primary antibody (the antibody of interest) diluted in blocking buffer, washing the membrane in washing buffer, blocking the membrane with a secondary antibody (which recognizes the primary antibody, e.g., an anti-human antibody) conjugated to an enzymatic substrate (e.g., horseradish peroxidase or alkaline phosphatase) or radioactive molecule (e.g., 32P or 125I) diluted in blocking buffer, washing the membrane in wash buffer, and detecting the presence of the antigen. One of skill in the art would be knowledgeable as to the parameters that can be modified to increase the signal detected and to reduce the background noise. For further discussion regarding western blot protocols see, e.g., Ausubel et al, eds, (1994), Current Protocols in Molecular Biology, Vol. 1, John Wiley & Sons, Inc., New York, section 10.8.1.

ELISAs comprise preparing antigen, coating the well of a 96 well microtiter plate with the antigen, adding the antibody of interest conjugated to a detectable compound such as an enzymatic substrate (e.g., horseradish peroxidase or alkaline phosphatase) to the well and incubating for a period of time, and detecting the presence of the antigen. In ELISAs the antibody of interest does not have to be conjugated to a detectable compound; instead, a second antibody (which recognizes the antibody of interest) conjugated to a detectable compound may be added to the well. Further, instead of coating the well with the antigen, the antibody may be coated to the well. In this case, a

second antibody conjugated to a detectable compound may be added following the addition of the antigen of interest to the coated well. One of skill in the art would be knowledgeable as to the parameters that can be modified to increase the signal detected as well as other variations of ELISAs known in the art. For further discussion regarding ELISAs see, e.g., Ausubel et al, eds, (1994), Current Protocols in Molecular Biology, Vol. 1, John Wiley & Sons, Inc., New York, section 11.2.1.

The binding affinity of an antibody to an antigen and the off-rate of an antibody-antigen interaction can be determined by competitive binding assays. One example of a competitive binding assay is a radioimmunoassay comprising the incubation of labeled antigen (e.g., 3H or 125I) with the antibody of interest in the presence of increasing amounts of unlabeled antigen, and the detection of the antibody bound to the labeled antigen. The affinity of the antibody of interest for a particular antigen and the binding off-rates can be determined from the data by scatchard plot analysis. Competition with a second antibody can also be determined using radioimmunoassays. In this case, the antigen is incubated with antibody of interest conjugated to a labeled compound (e.g., 3H or 125I) in the presence of increasing amounts of an unlabeled second antibody.

Antibodies of the invention may be characterized using immunocytochemisty methods on cells (e.g., mammalian cells, such as CHO cells) transfected with a vector enabling the expression of an antigen or with vector alone using techniques commonly known in the art. Antibodies that bind antigen transfected cells, but not vector-only transfected cells, are antigen specific.

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#### Therapeutic Uses

Table 1D also provides information regarding biological activities and preferred therapeutic uses (i.e. see, "Preferred Indications" column) for polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof). Table 1D also provides information regarding assays which may be used to test polynucleotides and polypeptides of the invention (including antibodies, agonists, and/or antagonists thereof) for the corresponding biological activities. The first column ("Gene No.") provides the gene number in the application for each clone identifier. The second column ("cDNA ATCC Deposit No:Z") provides the unique clone identifier for each clone as previously described and indicated in Table 1A, Table 1B, and Table 1C. The third column ("AA SEQ ID NO:Y") indicates the Sequence Listing SEQ ID Number for polypeptide sequences encoded by the corresponding cDNA clones (also as indicated in Table 1A, Table 1B, and Table 2). The fourth column ("Biological Activity") indicates a biological activity corresponding to the indicated polypeptides (or polynucleotides encoding said polypeptides). The fifth column ("Exemplary Activity Assay") further describes the corresponding biological activity and also provides information pertaining to the various types of

assays which may be performed to test, demonstrate, or quantify the corresponding biological activity.

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The present invention is further directed to antibody-based therapies which involve administering antibodies of the invention to an animal, preferably a mammal, and most preferably a human, patient for treating one or more of the disclosed diseases, disorders, or conditions. Therapeutic compounds of the invention include, but are not limited to, antibodies of the invention (including fragments, analogs and derivatives thereof as described herein) and nucleic acids encoding antibodies of the invention (including fragments, analogs and derivatives thereof and anti-idiotypic antibodies as described herein). The antibodies of the invention can be used to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate diseases, disorders or conditions associated with aberrant expression and/or activity of a polypeptide of the invention, including, but not limited to, hematopoietic and hematologic diseases and disorders. The treatment and/or prevention of hematopoietic and hematologic diseases and disorders associated with aberrant expression and/or activity of a polypeptide of the invention includes, but is not limited to, alleviating symptoms associated with hematopoietic and hematologic diseases and disorders. Antibodies of the invention may be provided in pharmaceutically acceptable compositions as known in the art or as described herein.

In a specific and preferred embodiment, the present invention is directed to antibodybased therapies which involve administering antibodies of the invention to an animal, preferably a mammal, and most preferably a human, patient for treating hematopoietic and hematologic diseases and disorders. Therapeutic compounds of the invention include, but are not limited to, antibodies of the invention (e.g., antibodies directed to the full length protein expressed on the cell surface of a mammalian cell; antibodies directed to an epitope of a polypeptide of the invention (such as, for example, a predicted linear epitope shown in Table 1B; or a conformational epitope, including fragments, analogs and derivatives thereof as described herein) and nucleic acids encoding antibodies of the invention (including fragments, analogs and derivatives thereof and anti-idiotypic antibodies as described herein). The antibodies of the invention can be used to detect, diagnose, prevent, treat, prognosticate, and/or ameliorate hematopoietic and hematologic diseases, disorders or conditions associated with aberrant expression and/or activity of a polypeptide of the invention. The treatment and/or prevention of hematopoietic and hematologic diseases, disorders or conditions associated with aberrant expression and/or activity of a polypeptide of the invention includes, but is not limited to, alleviating symptoms associated with those diseases, disorders or conditions. Antibodies of the invention may be provided in pharmaceutically acceptable compositions as known in the art or as described herein.

A summary of the ways in which the antibodies of the present invention may be used therapeutically includes binding polynucleotides or polypeptides of the present invention locally or systemically in the body or by direct cytotoxicity of the antibody, e.g. as mediated by complement

(CDC) or by effector cells (ADCC). Some of these approaches are described in more detail below. Armed with the teachings provided herein, one of ordinary skill in the art will know how to use the antibodies of the present invention for diagnostic, monitoring or therapeutic purposes without undue experimentation.

The antibodies of this invention may be advantageously utilized in combination with other monoclonal or chimeric antibodies, or with lymphokines or hematopoietic growth factors (such as, e.g., IL-2, IL-3 and IL-7), for example, which serve to increase the number or activity of effector cells which interact with the antibodies.

The antibodies of the invention may be administered alone or in combination with other types of treatments (e.g., radiation therapy, chemotherapy, hormonal therapy, immunotherapy and anti-tumor agents). Generally, administration of products of a species origin or species reactivity (in the case of antibodies) that is the same species as that of the patient is preferred. Thus, in a preferred embodiment, human antibodies, fragments derivatives, analogs, or nucleic acids, are administered to a human patient for therapy or prophylaxis.

It is preferred to use high affinity and/or potent *in vivo* inhibiting and/or neutralizing antibodies against polypeptides or polynucleotides of the present invention, fragments or regions thereof, for both immunoassays directed to and therapy of hematopoietic and hematologic diseases or disorders related to polynucleotides or polypeptides, including fragments thereof, of the present invention. Such antibodies, fragments, or regions, will preferably have an affinity for polynucleotides or polypeptides of the invention, including fragments thereof. Preferred binding affinities include those with a dissociation constant or Kd less than 5 X 10⁻² M, 10⁻² M, 5 X 10⁻³ M, 10⁻³ M, 5 X 10⁻⁴ M, 10⁻⁴ M, 5 X 10⁻⁵ M, 10⁻⁵ M, 5 X 10⁻⁶ M, 10⁻⁶ M, 5 X 10⁻¹⁷ M, 10⁻⁷ M, 5 X 10⁻⁸ M, 10⁻⁸ M, 5 X 10⁻¹⁹ M, 10⁻⁹ M, 5 X 10⁻¹⁰ M, 10⁻¹⁰ M, 5 X 10⁻¹¹ M, 10⁻¹¹ M, 5 X 10⁻¹² M, 10⁻¹³ M, 5 X 10⁻¹⁴ M, 10⁻¹⁴ M, 5 X 10⁻¹⁵ M, and 10⁻¹⁵ M.

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#### Gene Therapy

In a specific embodiment, nucleic acids comprising sequences encoding antibodies or functional derivatives thereof, are administered to treat, inhibit or prevent a hematopoietic and hematologic disease or disorder associated with aberrant expression and/or activity of a polypeptide of the invention, by way of gene therapy. Gene therapy refers to therapy performed by the administration to a subject of an expressed or expressible nucleic acid. In this embodiment of the invention, the nucleic acids produce their encoded protein that mediates a therapeutic effect.

Any of the methods for gene therapy available in the art can be used according to the present invention. Exemplary methods are described below.

For general reviews of the methods of gene therapy, see Goldspiel et al., Clinical Pharmacy 12:488-505 (1993); Wu and Wu, Biotherapy 3:87-95 (1991); Tolstoshev, Ann. Rev. Pharmacol. Toxicol. 32:573-596 (1993); Mulligan, Science 260:926-932 (1993); and Morgan and

Anderson, Ann. Rev. Biochem. 62:191-217 (1993); May, TIBTECH 11(5):155-215 (1993). Methods commonly known in the art of recombinant DNA technology which can be used are described in Ausubel et al. (eds.), Current Protocols in Molecular Biology, John Wiley & Sons, NY (1993); and Kriegler, Gene Transfer and Expression, A Laboratory Manual, Stockton Press, NY (1990).

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In a preferred embodiment, the compound comprises nucleic acid sequences encoding an antibody, said nucleic acid sequences being part of expression vectors that express the antibody or fragments or chimeric proteins or heavy or light chains thereof in a suitable host. In particular, such nucleic acid sequences have promoters operably linked to the antibody coding region, said promoter being inducible or constitutive, and, optionally, tissue-specific. In another particular embodiment, nucleic acid molecules are used in which the antibody coding sequences and any other desired sequences are flanked by regions that promote homologous recombination at a desired site in the genome, thus providing for intrachromosomal expression of the antibody encoding nucleic acids (Koller and Smithies, Proc. Natl. Acad. Sci. USA 86:8932-8935 (1989); Zijlstra et al., Nature 342:435-438 (1989). In specific embodiments, the expressed antibody molecule is a single chain antibody; alternatively, the nucleic acid sequences include sequences encoding both the heavy and light chains, or fragments thereof, of the antibody.

Delivery of the nucleic acids into a patient may be either direct, in which case the patient is directly exposed to the nucleic acid or nucleic acid-carrying vectors, or indirect, in which case, cells are first transformed with the nucleic acids in vitro, then transplanted into the patient. These two approaches are known, respectively, as *in vivo* or ex vivo gene therapy.

In a specific embodiment, the nucleic acid sequences are directly administered in vivo, where it is expressed to produce the encoded product. This can be accomplished by any of numerous methods known in the art, e.g., by constructing them as part of an appropriate nucleic acid expression vector and administering it so that they become intracellular, e.g., by infection using defective or attenuated retrovirals or other viral vectors (see U.S. Patent No. 4,980,286), or by direct injection of naked DNA, or by use of microparticle bombardment (e.g., a gene gun; Biolistic, Dupont), or coating with lipids or cell-surface receptors or transfecting agents, encapsulation in liposomes, microparticles, or microcapsules, or by administering them in linkage to a peptide which is known to enter the nucleus, by administering it in linkage to a ligand subject to receptor-mediated endocytosis (see, e.g., Wu and Wu, J. Biol. Chem. 262:4429-4432 (1987)) (which can be used to target cell types specifically expressing the receptors), etc. In another embodiment, nucleic acid-ligand complexes can be formed in which the ligand comprises a fusogenic viral peptide to disrupt endosomes, allowing the nucleic acid to avoid lysosomal degradation. In yet another embodiment, the nucleic acid can be targeted in vivo for cell specific uptake and expression, by targeting a specific receptor (see, e.g., PCT Publications WO 92/06180; WO 92/22635; WO92/20316; WO93/14188, WO 93/20221). Alternatively, the nucleic acid can

be introduced intracellularly and incorporated within host cell DNA for expression, by homologous recombination (Koller and Smithies, Proc. Natl. Acad. Sci. USA 86:8932-8935 (1989); Zijlstra et al., Nature 342:435-438 (1989)).

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In a specific embodiment, viral vectors that contain nucleic acid sequences encoding an antibody of the invention are used. For example, a retroviral vector can be used (see Miller et al., Meth. Enzymol. 217:581-599 (1993)). These retroviral vectors contain the components necessary for the correct packaging of the viral genome and integration into the host cell DNA. The nucleic acid sequences encoding the antibody to be used in gene therapy are cloned into one or more vectors, which facilitates delivery of the gene into a patient. More detail about retroviral vectors can be found in Boesen et al., Biotherapy 6:291-302 (1994), which describes the use of a retroviral vector to deliver the mdr1 gene to hematopoietic stem cells in order to make the stem cells more resistant to chemotherapy. Other references illustrating the use of retroviral vectors in gene therapy are: Clowes et al., J. Clin. Invest. 93:644-651 (1994); Kiem et al., Blood 83:1467-1473 (1994); Salmons and Gunzberg, Human Gene Therapy 4:129-141 (1993); and Grossman and Wilson, Curr. Opin. in Genetics and Devel. 3:110-114 (1993).

Adenoviruses are other viral vectors that can be used in gene therapy. Adenoviruses are especially attractive vehicles for delivering genes to respiratory epithelia. Adenoviruses naturally infect respiratory epithelia where they cause a mild disease. Other targets for adenovirus-based delivery systems are liver, the central nervous system, endothelial cells, and muscle. Adenoviruses have the advantage of being capable of infecting non-dividing cells. Kozarsky and Wilson, Current Opinion in Genetics and Development 3:499-503 (1993) present a review of adenovirus-based gene therapy. Bout et al., Human Gene Therapy 5:3-10 (1994) demonstrated the use of adenovirus vectors to transfer genes to the respiratory epithelia of rhesus monkeys. Other instances of the use of adenoviruses in gene therapy can be found in Rosenfeld et al., Science 252:431-434 (1991); Rosenfeld et al., Cell 68:143- 155 (1992); Mastrangeli et al., J. Clin. Invest. 91:225-234 (1993); PCT Publication WO94/12649; and Wang, et al., Gene Therapy 2:775-783 (1995). In a preferred embodiment, adenovirus vectors are used.

Adeno-associated virus (AAV) has also been proposed for use in gene therapy (Walsh et al., Proc. Soc. Exp. Biol. Med. 204:289-300 (1993); U.S. Patent No. 5,436,146).

Another approach to gene therapy involves transferring a gene to cells in tissue culture by such methods as electroporation, lipofection, calcium phosphate mediated transfection, or viral infection. Usually, the method of transfer includes the transfer of a selectable marker to the cells. The cells are then placed under selection to isolate those cells that have taken up and are expressing the transferred gene. Those cells are then delivered to a patient.

In this embodiment, the nucleic acid is introduced into a cell prior to administration in vivo of the resulting recombinant cell. Such introduction can be carried out by any method known in the art, including but not limited to transfection, electroporation, microinjection, infection with a

viral or bacteriophage vector containing the nucleic acid sequences, cell fusion, chromosome-mediated gene transfer, microcell-mediated gene transfer, spheroplast fusion, etc. Numerous techniques are known in the art for the introduction of foreign genes into cells (see, e.g., Loeffler and Behr, Meth. Enzymol. 217:599-618 (1993); Cohen et al., Meth. Enzymol. 217:618-644 (1993); Cline, Pharmac. Ther. 29:69-92m (1985) and may be used in accordance with the present invention, provided that the necessary developmental and physiological functions of the recipient cells are not disrupted. The technique should provide for the stable transfer of the nucleic acid to the cell, so that the nucleic acid is expressible by the cell and preferably heritable and expressible by its cell progeny.

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The resulting recombinant cells can be delivered to a patient by various methods known in the art. Recombinant blood cells (e.g., hematopoietic stem or progenitor cells) are preferably administered intravenously. The amount of cells envisioned for use depends on the desired effect, patient state, etc., and can be determined by one skilled in the art.

Cells into which a nucleic acid can be introduced for purposes of gene therapy encompass any desired, available cell type, and include but are not limited to epithelial cells, endothelial cells, keratinocytes, fibroblasts, muscle cells, hepatocytes; blood cells such as T lymphocytes, B lymphocytes, monocytes, macrophages, neutrophils, eosinophils, megakaryocytes, granulocytes; various stem or progenitor cells, in particular hematopoietic stem or progenitor cells, e.g., as obtained from bone marrow, umbilical cord blood, peripheral blood, fetal liver, etc.

In a preferred embodiment, the cell used for gene therapy is autologous to the patient.

In an embodiment in which recombinant cells are used in gene therapy, nucleic acid sequences encoding an antibody are introduced into the cells such that they are expressible by the cells or their progeny, and the recombinant cells are then administered *in vivo* for therapeutic effect. In a specific embodiment, stem or progenitor cells are used. Any stem and/or progenitor cells which can be isolated and maintained in vitro can potentially be used in accordance with this embodiment of the present invention (see e.g. PCT Publication WO 94/08598; Stemple and Anderson, Cell 71:973-985 (1992); Rheinwald, Meth. Cell Bio. 21A:229 (1980); and Pittelkow and Scott, Mayo Clinic Proc. 61:771 (1986)).

In a specific embodiment, the nucleic acid to be introduced for purposes of gene therapy comprises an inducible promoter operably linked to the coding region, such that expression of the nucleic acid is controllable by the presence or absence of an appropriate inducer of transcription.

### Demonstration of Therapeutic or Prophylactic Activity

The compounds or pharmaceutical compositions of the invention are preferably tested in vitro, and then *in vivo* for the desired therapeutic or prophylactic activity, prior to use in humans. For example, in vitro assays to demonstrate the therapeutic or prophylactic utility of a compound or pharmaceutical composition include, the effect of a compound on a cell line or a patient tissue

sample. The effect of the compound or composition on the cell line and/or tissue sample can be determined utilizing techniques known to those of skill in the art including, but not limited to, rosette formation assays and cell lysis assays. In accordance with the invention, in vitro assays which can be used to determine whether administration of a specific compound is indicated, include in vitro cell culture assays in which a patient tissue sample is grown in culture, and exposed to or otherwise administered a compound, and the effect of such compound upon the tissue sample is observed.

### Therapeutic/Prophylactic Administration and Composition

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The invention provides methods of treatment, inhibition and prophylaxis by administration to a subject of an effective amount of a compound or pharmaceutical composition of the invention, preferably a polypeptide or antibody of the invention. In a preferred embodiment, the compound is substantially purified (e.g., substantially free from substances that limit its effect or produce undesired side-effects). The subject is preferably an animal, including but not limited to animals such as cows, pigs, horses, chickens, cats, dogs, etc., and is preferably a mammal, and most preferably human.

Formulations and methods of administration that can be employed when the compound comprises a nucleic acid or an immunoglobulin are described above; additional appropriate formulations and routes of administration can be selected from among those described herein below.

Various delivery systems are known and can be used to administer a compound of the invention, e.g., encapsulation in liposomes, microparticles, microcapsules, recombinant cells capable of expressing the compound, receptor-mediated endocytosis (see, e.g., Wu and Wu, J. Biol. Chem. 262:4429-4432 (1987)), construction of a nucleic acid as part of a retroviral or other vector, etc. Methods of introduction include but are not limited to intradermal, intramuscular, intraperitoneal, intravenous, subcutaneous, intranasal, epidural, and oral routes. The compounds or compositions may be administered by any convenient route, for example by infusion or bolus injection, by absorption through epithelial or mucocutaneous linings (e.g., oral mucosa, rectal and intestinal mucosa, etc.) and may be administered together with other biologically active agents. Administration can be systemic or local. In addition, it may be desirable to introduce the pharmaceutical compounds or compositions of the invention into the central nervous system by any suitable route, including intraventricular and intrathecal injection; intraventricular injection may be facilitated by an intraventricular catheter, for example, attached to a reservoir, such as an Ommaya reservoir. Pulmonary administration can also be employed, e.g., by use of an inhaler or nebulizer, and formulation with an aerosolizing agent.

In a specific embodiment, it may be desirable to administer the pharmaceutical compounds or compositions of the invention locally to the area in need of treatment; this may be

achieved by, for example, and not by way of limitation, local infusion during surgery, topical application, e.g., in conjunction with a wound dressing after surgery, by injection, by means of a catheter, by means of a suppository, or by means of an implant, said implant being of a porous, non-porous, or gelatinous material, including membranes, such as sialastic membranes, or fibers. Preferably, when administering a protein, including an antibody, of the invention, care must be taken to use materials to which the protein does not absorb.

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In another embodiment, the compound or composition can be delivered in a vesicle, in particular a liposome (see Langer, Science 249:1527-1533 (1990); Treat et al., in Liposomes in the Therapy of Infectious Disease and Cancer, Lopez-Berestein and Fidler (eds.), Liss, New York, pp. 353-365 (1989); Lopez-Berestein, ibid., pp. 317-327; see generally ibid.)

In yet another embodiment, the compound or composition can be delivered in a controlled release system. In one embodiment, a pump may be used (see Langer, *supra*; Sefton, CRC Crit. Ref. Biomed. Eng. 14:201 (1987); Buchwald et al., Surgery 88:507 (1980); Saudek et al., N. Engl. J. Med. 321:574 (1989)). In another embodiment, polymeric materials can be used (see Medical Applications of Controlled Release, Langer and Wise (eds.), CRC Pres., Boca Raton, Florida (1974); Controlled Drug Bioavailability, Drug Product Design and Performance, Smolen and Ball (eds.), Wiley, New York (1984); Ranger and Peppas, J., Macromol. Sci. Rev. Macromol. Chem. 23:61 (1983); see also Levy et al., Science 228:190 (1985); During et al., Ann. Neurol. 25:351 (1989); Howard et al., J.Neurosurg. 71:105 (1989)). In yet another embodiment, a controlled release system can be placed in proximity of the therapeutic target, e.g., the brain, thus requiring only a fraction of the systemic dose (see, e.g., Goodson, in Medical Applications of Controlled Release, *supra*, vol. 2, pp. 115-138 (1984)).

Other controlled release systems are discussed in the review by Langer (Science 249:1527-1533 (1990)).

In a specific embodiment where the compound of the invention is a nucleic acid encoding a protein, the nucleic acid can be administered *in vivo* to promote expression of its encoded protein, by constructing it as part of an appropriate nucleic acid expression vector and administering it so that it becomes intracellular, e.g., by use of a retroviral vector (see U.S. Patent No. 4,980,286), or by direct injection, or by use of microparticle bombardment (e.g., a gene gun; Biolistic, Dupont), or coating with lipids or cell-surface receptors or transfecting agents, or by administering it in linkage to a homeobox-like peptide which is known to enter the nucleus (see e.g., Joliot et al., Proc. Natl. Acad. Sci. USA 88:1864-1868 (1991)), etc. Alternatively, a nucleic acid can be introduced intracellularly and incorporated within host cell DNA for expression, by homologous recombination.

The present invention also provides pharmaceutical compositions. Such compositions comprise a therapeutically effective amount of a compound, and a pharmaceutically acceptable carrier. In a specific embodiment, the term "pharmaceutically acceptable" means approved by a

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regulatory agency of the Federal or a state government or listed in the U.S. Pharmacopeia or other generally recognized pharmacopeia for use in animals, and mor particularly in humans. The term "carrier" refers to a diluent, adjuvant, excipient, or vehicle with which the therapeutic is administered. Such pharmaceutical carriers can be sterile liquids, such as water and oils, including those of petroleum, animal, vegetable or synthetic origin, such as peanut oil, soybean oil, mineral oil, sesame oil and the like. Water is a preferred carrier when the pharmaceutical composition is administered intravenously. Saline solutions and aqueous dextrose and glycerol solutions can also be employed as liquid carriers, particularly for injectable solutions. Suitable pharmaceutical excipients include starch, glucose, lactose, sucrose, gelatin, malt, rice, flour, chalk, silica gel, sodium stearate, glycerol monostearate, talc, sodium chloride, dried skim milk, glycerol, propylene, glycol, water, ethanol and the like. The composition, if desired, can also contain minor amounts of wetting or emulsifying agents, or pH buffering agents. These compositions can take the form of solutions, suspensions, emulsion, tablets, pills, capsules, powders, sustained-release formulations and the like. The composition can be formulated as a suppository, with traditional binders and carriers such as triglycerides. Oral formulation can include standard carriers such as pharmaceutical grades of mannitol, lactose, starch, magnesium stearate, sodium saccharine, cellulose, magnesium carbonate, etc. Examples of suitable pharmaceutical carriers are described in "Remington's Pharmaceutical Sciences" by E.W. Martin. Such compositions will contain a therapeutically effective amount of the compound, preferably in purified form, together with a suitable amount of carrier so as to provide the form for proper administration to the patient. The formulation should suit the mode of administration.

In a preferred embodiment, the composition is formulated in accordance with routine procedures as a pharmaceutical composition adapted for intravenous administration to human beings. Typically, compositions for intravenous administration are solutions in sterile isotonic aqueous buffer. Where necessary, the composition may also include a solubilizing agent and a local anesthetic such as lignocaine to ease pain at the site of the injection. Generally, the ingredients are supplied either separately or mixed together in unit dosage form, for example, as a dry lyophilized powder or water free concentrate in a hermetically sealed container such as an ampoule or sachette indicating the quantity of active agent. Where the composition is to be administered by infusion, it can be dispensed with an infusion bottle containing sterile pharmaceutical grade water or saline. Where the composition is administered by injection, an ampoule of sterile water for injection or saline can be provided so that the ingredients may be mixed prior to administration.

The compounds of the invention can be formulated as neutral or salt forms. Pharmaceutically acceptable salts include those formed with anions such as those derived from hydrochloric, phosphoric, acetic, oxalic, tartaric acids, etc., and those formed with cations such as

those derived from sodium, potassium, ammonium, calcium, ferric hydroxides, isopropylamine, triethylamine, 2-ethylamino ethanol, histidine, procaine, etc.

The amount of the compound of the invention which will be effective in the treatment, inhibition and prevention of a disease or disorder associated with aberrant expression and/or activity of a polypeptide of the invention can be determined by standard clinical techniques. In addition, in vitro assays may optionally be employed to help identify optimal dosage ranges. The precise dose to be employed in the formulation will also depend on the route of administration, and the seriousness of the disease or disorder, and should be decided according to the judgment of the practitioner and each patient's circumstances. Effective doses may be extrapolated from dose-response curves derived from in vitro or animal model test systems.

For antibodies, the dosage administered to a patient is typically 0.1 mg/kg to 100 mg/kg of the patient's body weight. Preferably, the dosage administered to a patient is between 0.1 mg/kg and 20 mg/kg of the patient's body weight, more preferably 1 mg/kg to 10 mg/kg of the patient's body weight. Generally, human antibodies have a longer half-life within the human body than antibodies from other species due to the immune response to the foreign polypeptides. Thus, lower dosages of human antibodies and less frequent administration is often possible. Further, the dosage and frequency of administration of antibodies of the invention may be reduced by enhancing uptake and tissue penetration (e.g., into the brain) of the antibodies by modifications such as, for example, lipidation.

The invention also provides a pharmaceutical pack or kit comprising one or more containers filled with one or more of the ingredients of the pharmaceutical compositions of the invention. Optionally associated with such container(s) can be a notice in the form prescribed by a governmental agency regulating the manufacture, use or sale of pharmaceuticals or biological products, which notice reflects approval by the agency of manufacture, use or sale for human administration.

# Diagnosis and Imaging

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Labeled antibodies, and derivatives and analogs thereof, which specifically bind to a polypeptide of interest can be used for diagnostic purposes to detect, diagnose, prognosticate, or monitor hematopoietic and hematologic diseases, disorders, and/or conditions associated with the aberrant expression and/or activity of a polypeptide of the invention. The invention provides for the detection of aberrant expression of a polypeptide of interest, comprising (a) assaying the expression of the polypeptide of interest in cells or body fluid of an individual using one or more antibodies specific to the polypeptide interest and (b) comparing the level of gene expression with a standard gene expression level, whereby an increase or decrease in the assayed polypeptide gene expression level compared to the standard expression level is indicative of aberrant expression.

The invention provides a diagnostic assay for diagnosing a hematopoietic and hematologic disease or disorder, comprising (a) assaying the expression of the polypeptide of interest in cells or body fluid of an individual using one or more antibodies specific to the polypeptide interest and (b) comparing the level of gene expression with a standard gene expression level, whereby an increase or decrease in the assayed polypeptide gene expression level compared to the standard expression level is indicative of a particular hematopoietic and hematologic disease or disorder. With respect to hematopoietic cancers, the presence of a relatively high amount of transcript in biopsied tissue from an individual may indicate a predisposition for the development of the disease, or may provide a means for detecting the disease prior to the appearance of actual clinical symptoms. A more definitive diagnosis of this type may allow health professionals to employ preventative measures or aggressive treatment earlier thereby preventing the development or further progression of the hematopoietic cancer.

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Antibodies of the invention can be used to assay protein levels in a biological sample using classical immunohistological methods known to those of skill in the art (e.g., see Jalkanen et al., J. Cell. Biol. 101:976-985 (1985); Jalkanen et al., J. Cell. Biol. 105:3087-3096 (1987)). Other antibody-based methods useful for detecting protein gene expression include immunoassays, such as the enzyme linked immunosorbent assay (ELISA) and the radioimmunoassay (RIA). Suitable antibody assay labels are known in the art and include enzyme labels, such as, glucose oxidase; radioisotopes, such as iodine (125I, 121I), carbon (14C), sulfur (35S), tritium (3H), indium (112In), and technetium (99Tc); luminescent labels, such as luminol; and fluorescent labels, such as fluorescein and rhodamine, and biotin.

One facet of the invention is the detection and diagnosis of a disease or disorder associated with aberrant expression of a polypeptide of interest in an animal, preferably a mammal and most preferably a human. In one embodiment, diagnosis comprises: a) administering (for example, parenterally, subcutaneously, or intraperitoneally) to a subject an effective amount of a labeled molecule which specifically binds to the polypeptide of interest; b) waiting for a time interval following the administering for permitting the labeled molecule to preferentially concentrate at sites in the subject where the polypeptide is expressed (and for unbound labeled molecule to be cleared to background level); c) determining background level; and d) detecting the labeled molecule in the subject, such that detection of labeled molecule above the background level indicates that the subject has a particular disease or disorder associated with aberrant expression of the polypeptide of interest. Background level can be determined by various methods including, comparing the amount of labeled molecule detected to a standard value previously determined for a particular system.

It will be understood in the art that the size of the subject and the imaging system used will determine the quantity of imaging moiety needed to produce diagnostic images. In the case of a radioisotope moiety, for a human subject, the quantity of radioactivity injected will normally range

from about 5 to 20 millicuries of 99mTc. The labeled antibody or antibody fragment will then preferentially accumulate at the location of cells which contain the specific protein. *In vivo* tumor imaging is described in S.W. Burchiel et al., "Immunopharmacokinetics of Radiolabeled Antibodies and Their Fragments." (Chapter 13 in Tumor Imaging: The Radiochemical Detection of Cancer, S.W. Burchiel and B. A. Rhodes, eds., Masson Publishing Inc. (1982)).

Depending on several variables, including the type of label used and the mode of administration, the time interval following the administration for permitting the labeled molecule to preferentially concentrate at sites in the subject and for unbound labeled molecule to be cleared to background level is 6 to 48 hours or 6 to 24 hours or 6 to 12 hours. In another embodiment the time interval following administration is 5 to 20 days or 5 to 10 days.

In an embodiment, monitoring of the disease or disorder is carried out by repeating the method for diagnosing the disease or disease, for example, one month after initial diagnosis, six months after initial diagnosis, one year after initial diagnosis, etc.

Presence of the labeled molecule can be detected in the patient using methods known in the art for *in vivo* scanning. These methods depend upon the type of label used. Skilled artisans will be able to determine the appropriate method for detecting a particular label. Methods and devices that may be used in the diagnostic methods of the invention include, but are not limited to, computed tomography (CT), whole body scan such as position emission tomography (PET), magnetic resonance imaging (MRI), and sonography.

In a specific embodiment, the molecule is labeled with a radioisotope and is detected in the patient using a radiation responsive surgical instrument (Thurston et al., U.S. Patent No. 5,441,050). In another embodiment, the molecule is labeled with a fluorescent compound and is detected in the patient using a fluorescence responsive scanning instrument. In another embodiment, the molecule is labeled with a positron emitting metal and is detected in the patent using positron emission-tomography. In yet another embodiment, the molecule is labeled with a paramagnetic label and is detected in a patient using magnetic resonance imaging (MRI).

Kits

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The present invention provides kits that can be used in the above methods. In one embodiment, a kit comprises an antibody of the invention, preferably a purified antibody, in one or more containers. In a specific embodiment, the kits of the present invention contain a substantially isolated polypeptide comprising an epitope which is specifically immunoreactive with an antibody included in the kit. Preferably, the kits of the present invention further comprise a control antibody which does not react with the polypeptide of interest. In another specific embodiment, the kits of the present invention contain a means for detecting the binding of an antibody to a polypeptide of interest (e.g., the antibody may be conjugated to a detectable substrate such as a fluorescent compound, an enzymatic substrate, a radioactive compound or a luminescent

compound, or a second antibody which recognizes the first antibody may be conjugated to a detectable substrate).

In another specific embodiment of the present invention, the kit is a diagnostic kit for use in screening serum containing antibodies specific against proliferative and/or cancerous polynucleotides and polypeptides. Such a kit may include a control antibody that does not react with the polypeptide of interest. Such a kit may include a substantially isolated polypeptide antigen comprising an epitope which is specifically immunoreactive with at least one antipolypeptide antigen antibody. Further, such a kit includes means for detecting the binding of said antibody to the antigen (e.g., the antibody may be conjugated to a fluorescent compound such as fluorescein or rhodamine which can be detected by flow cytometry). In specific embodiments, the kit may include a recombinantly produced or chemically synthesized polypeptide antigen. The polypeptide antigen of the kit may also be attached to a solid support.

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In a more specific embodiment the detecting means of the above-described kit includes a solid support to which said polypeptide antigen is attached. Such a kit may also include a non-attached reporter-labeled anti-human antibody. In this embodiment, binding of the antibody to the polypeptide antigen can be detected by binding of the said reporter-labeled antibody.

In an additional embodiment, the invention includes a diagnostic kit for use in screening serum containing antigens of the polypeptide of the invention. The diagnostic kit includes a substantially isolated antibody specifically immunoreactive with polypeptide or polynucleotide antigens, and means for detecting the binding of the polynucleotide or polypeptide antigen to the antibody. In one embodiment, the antibody is attached to a solid support. In a specific embodiment, the antibody may be a monoclonal antibody. The detecting means of the kit may include a second, labeled monoclonal antibody. Alternatively, or in addition, the detecting means may include a labeled, competing antigen.

In one diagnostic configuration, test serum is reacted with a solid phase reagent having a surface-bound antigen obtained by the methods of the present invention. After binding with specific antigen antibody to the reagent and removing unbound serum components by washing, the reagent is reacted with reporter-labeled anti-human antibody to bind reporter to the reagent in proportion to the amount of bound anti-antigen antibody on the solid support. The reagent is again washed to remove unbound labeled antibody, and the amount of reporter associated with the reagent is determined. Typically, the reporter is an enzyme which is detected by incubating the solid phase in the presence of a suitable fluorometric, luminescent or colorimetric substrate (Sigma, St. Louis, MO).

The solid surface reagent in the above assay is prepared by known techniques for attaching protein material to solid support material, such as polymeric beads, dip sticks, 96-well plate or filter material. These attachment methods generally include non-specific adsorption of the protein to the support or covalent attachment of the protein, typically through a free amine group, to a

chemically reactive group on the solid support, such as an activated carboxyl, hydroxyl, or aldehyde group. Alternatively, streptavidin coated plates can be used in conjunction with biotinylated antigen(s).

Thus, the invention provides an assay system or kit for carrying out this diagnostic method. The kit generally includes a support with surface-bound recombinant antigens, and a reporter-labeled anti-human antibody for detecting surface-bound anti-antigen antibody.

### Uses of the Polynucleotides

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Each of the polynucleotides identified herein can be used in numerous ways as reagents. The following description should be considered exemplary and utilizes known techniques.

The polynucleotides of the present invention are useful for chromosome identification. There exists an ongoing need to identify new chromosome markers, since few chromosome marking reagents, based on actual sequence data (repeat polymorphisms), are presently available. Each sequence is specifically targeted to and can hybridize with a particular location on an individual human chromosome, thus each polynucleotide of the present invention can routinely be used as a chromosome marker using techniques known in the art. Table 1B.1, column 8 provides the chromosome location of some of the polynucleotides of the invention.

Briefly, sequences can be mapped to chromosomes by preparing PCR primers (preferably at least 15 bp (e.g., 15-25 bp) from the sequences shown in SEQ ID NO:X. Primers can optionally be selected using computer analysis so that primers do not span more than one predicted exon in the genomic DNA. These primers are then used for PCR screening of somatic cell hybrids containing individual human chromosomes. Only those hybrids containing the human gene corresponding to SEQ ID NO:X will yield an amplified fragment.

Similarly, somatic hybrids provide a rapid method of PCR mapping the polynucleotides to particular chromosomes. Three or more clones can be assigned per day using a single thermal cycler. Moreover, sublocalization of the polynucleotides can be achieved with panels of specific chromosome fragments. Other gene mapping strategies that can be used include in situ hybridization, prescreening with labeled flow-sorted chromosomes, preselection by hybridization to construct chromosome specific-cDNA libraries, and computer mapping techniques (See, e.g., Shuler, Trends Biotechnol 16:456-459 (1998) which is hereby incorporated by reference in its entirety).

Precise chromosomal location of the polynucleotides can also be achieved using fluorescence in situ hybridization (FISH) of a metaphase chromosomal spread. This technique uses polynucleotides as short as 500 or 600 bases; however, polynucleotides 2,000-4,000 bp are preferred. For a review of this technique, see Verma et al., "Human Chromosomes: a Manual of Basic Techniques," Pergamon Press, New York (1988).

For chromosome mapping, the polynucleotides can be used individually (to mark a single chromosome or a single site on that chromosome) or in panels (for marking multiple sites and/or multiple chromosomes).

Thus, the present invention also provides a method for chromosomal localization which involves (a) preparing PCR primers from the polynucleotide sequences in Table 1B and/or Table 2 and SEQ ID NO:X and (b) screening somatic cell hybrids containing individual chromosomes.

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The polynucleotides of the present invention would likewise be useful for radiation hybrid mapping, HAPPY mapping, and long range restriction mapping. For a review of these techniques and others known in the art, see, e.g. Dear, "Genome Mapping: A Practical Approach," IRL Press at Oxford University Press, London (1997); Aydin, J. Mol. Med. 77:691-694 (1999); Hacia et al., Mol. Psychiatry 3:483-492 (1998); Herrick et al., Chromosome Res. 7:409-423 (1999); Hamilton et al., Methods Cell Biol. 62:265-280 (2000); and/or Ott, J. Hered. 90:68-70 (1999) each of which is hereby incorporated by reference in its entirety.

Once a polynucleotide has been mapped to a precise chromosomal location, the physical position of the polynucleotide can be used in linkage analysis. Linkage analysis establishes coinheritance between a chromosomal location and presentation of a particular disease. (Disease mapping data are found, for example, in V. McKusick, Mendelian Inheritance in Man (available on line through Johns Hopkins University Welch Medical Library)). Column 9 of Table 1B.1 provides an OMIM reference identification number of diseases associated with the cytologic band disclosed in column 8 of Table 1B.1, as determined using techniques described herein and by reference to Table 5. Assuming 1 megabase mapping resolution and one gene per 20 kb, a cDNA precisely localized to a chromosomal region associated with the disease could be one of 50-500 potential causative genes.

Thus, once coinheritance is established, differences in a polynucleotide of the invention and the corresponding gene between affected and unaffected individuals can be examined. First, visible structural alterations in the chromosomes, such as deletions or translocations, are examined in chromosome spreads or by PCR. If no structural alterations exist, the presence of point mutations are ascertained. Mutations observed in some or all affected individuals, but not in normal individuals, indicates that the mutation may cause the disease. However, complete sequencing of the polypeptide and the corresponding gene from several normal individuals is required to distinguish the mutation from a polymorphism. If a new polymorphism is identified, this polymorphic polypeptide can be used for further linkage analysis.

Furthermore, increased or decreased expression of the gene in affected individuals as compared to unaffected individuals can be assessed using the polynucleotides of the invention. Any of these alterations (altered expression, chromosomal rearrangement, or mutation) can be used as a diagnostic or prognostic marker. Diagnostic and prognostic methods, kits and reagents encompassed by the present invention are briefly described below and more thoroughly elsewhere

herein (see e.g., the sections labeled "Antibodies", "Diagnostic Assays", and "Methods for Detecting Diseases").

Thus, the invention also provides a diagnostic method useful during diagnosis of a disorder, involving measuring the expression level of polynucleotides of the present invention in cells or body fluid from an individual and comparing the measured gene expression level with a standard level of polynucleotide expression level, whereby an increase or decrease in the gene expression level compared to the standard is indicative of a disorder. Additional non-limiting examples of diagnostic methods encompassed by the present invention are more thoroughly described elsewhere herein (see, e.g., Example 12).

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In still another embodiment, the invention includes a kit for analyzing samples for the presence of proliferative and/or cancerous polynucleotides derived from a test subject. In a general embodiment, the kit includes at least one polynucleotide probe containing a nucleotide sequence that will specifically hybridize with a polynucleotide of the invention and a suitable container. In a specific embodiment, the kit includes two polynucleotide probes defining an internal region of the polynucleotide of the invention, where each probe has one strand containing a 31'mer-end internal to the region. In a further embodiment, the probes may be useful as primers for polymerase chain reaction amplification.

Where a diagnosis of a related disorder, including, for example, diagnosis of a tumor, has already been made according to conventional methods, the present invention is useful as a prognostic indicator, whereby patients exhibiting enhanced or depressed polynucleotide of the invention expression will experience a worse clinical outcome relative to patients expressing the gene at a level nearer the standard level.

By "measuring the expression level of polynucleotides of the invention" is intended qualitatively or quantitatively measuring or estimating the level of the polypeptide of the invention or the level of the mRNA encoding the polypeptide of the invention in a first biological sample either directly (e.g., by determining or estimating absolute protein level or mRNA level) or relatively (e.g., by comparing to the polypeptide level or mRNA level in a second biological sample). Preferably, the polypeptide level or mRNA level in the first biological sample is measured or estimated and compared to a standard polypeptide level or mRNA level, the standard being taken from a second biological sample obtained from an individual not having the related disorder or being determined by averaging levels from a population of individuals not having a related disorder. As will be appreciated in the art, once a standard polypeptide level or mRNA level is known, it can be used repeatedly as a standard for comparison.

By "biological sample" is intended any biological sample obtained from an individual, body fluid, cell line, tissue culture, or other source which contains polypeptide of the present invention or the corresponding mRNA. As indicated, biological samples include body fluids (such as semen, lymph, vaginal pool, sera, plasma, urine, synovial fluid and spinal fluid) which contain

the polypeptide of the present invention, and tissue sources found to express the polypeptide of the present invention. Methods for obtaining tissue biopsies and body fluids from mammals are well known in the art. Where the biological sample is to include mRNA, a tissue biopsy is the preferred source.

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The method(s) provided above may preferably be applied in a diagnostic method and/or kits in which polynucleotides and/or polypeptides of the invention are attached to a solid support. In one exemplary method, the support may be a "gene chip" or a "biological chip" as described in US Patents 5,837,832, 5,874,219, and 5,856,174. Further, such a gene chip with polynucleotides of the invention attached may be used to identify polymorphisms between the isolated polynucleotide sequences of the invention, with polynucleotides isolated from a test subject. The knowledge of such polymorphisms (i.e. their location, as well as, their existence) would be beneficial in identifying disease loci for many disorders, such as for example, in neural disorders, immune system disorders, muscular disorders, reproductive disorders, gastrointestinal disorders, pulmonary disorders, digestive disorders, metabolic disorders, cardiovascular disorders, renal disorders, proliferative disorders, and/or cancerous diseases and conditions. Such a method is described in US Patents 5,858,659 and 5,856,104. The US Patents referenced supra are hereby incorporated by reference in their entirety herein.

The present invention encompasses polynucleotides of the present invention that are chemically synthesized, or reproduced as peptide nucleic acids (PNA), or according to other methods known in the art. The use of PNAs would serve as the preferred form if the polynucleotides of the invention are incorporated onto a solid support, or gene chip. For the purposes of the present invention, a peptide nucleic acid (PNA) is a polyamide type of DNA analog and the monomeric units for adenine, guanine, thymine and cytosine are available commercially (Perceptive Biosystems). Certain components of DNA, such as phosphorus, phosphorus oxides, or deoxyribose derivatives, are not present in PNAs. As disclosed by Nielsen et al., Science 254, 1497 (1991); and Egholm et al., Nature 365, 666 (1993), PNAs bind specifically and tightly to complementary DNA strands and are not degraded by nucleases. In fact, PNA binds more strongly to DNA than DNA itself does. This is probably because there is no electrostatic repulsion between the two strands, and also the polyamide backbone is more flexible. Because of this, PNA/DNA duplexes bind under a wider range of stringency conditions than DNA/DNA duplexes, making it easier to perform multiplex hybridization. Smaller probes can be used than with DNA due to the strong binding. In addition, it is more likely that single base mismatches can be determined with PNA/DNA hybridization because a single mismatch in a PNA/DNA 15-mer lowers the melting point (T.sub.m) by 8°-20° C, vs. 4°-16° C for the DNA/DNA 15-mer duplex. Also, the absence of charge groups in PNA means that hybridization can be done at low ionic strengths and reduce possible interference by salt during the analysis.

The compounds of the present invention have uses which include, but are not limited to, detecting cancer in mammals. In particular the invention is useful during diagnosis of pathological cell proliferative neoplasias which include, but are not limited to: acute myelogenous leukemias including acute monocytic leukemia, acute myeloblastic leukemia, acute promyelocytic leukemia, acute myelomonocytic leukemia, acute erythroleukemia, acute megakaryocytic leukemia, and acute undifferentiated leukemia, etc.; and chronic myelogenous leukemias including chronic myelomonocytic leukemia, chronic granulocytic leukemia, etc. Preferred mammals include monkeys, apes, cats, dogs, cows, pigs, horses, rabbits and humans. Particularly preferred are humans.

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Pathological cell proliferative disorders are often associated with inappropriate activation of proto-oncogenes. (Gelmann, E. P. et al., "The Etiology of Acute Leukemia: Molecular Genetics and Viral Oncology," in Neoplastic Diseases of the Blood, Vol 1., Wiernik, P. H. et al. eds., 161-182 (1985)). Neoplasias are now believed to result from the qualitative alteration of a normal cellular gene product, or from the quantitative modification of gene expression by insertion into the chromosome of a viral sequence, by chromosomal translocation of a gene to a more actively transcribed region, or by some other mechanism. (Gelmann et al., *supra*) It is likely that mutated or altered expression of specific genes is involved in the pathogenesis of some leukemias, among other tissues and cell types. (Gelmann et al., *supra*) Indeed, the human counterparts of the oncogenes involved in some animal neoplasias have been amplified or translocated in some cases of human leukemia and carcinoma. (Gelmann et al., *supra*)

For example, c-myc expression is highly amplified in the non-lymphocytic leukemia cell line HL-60. When HL-60 cells are chemically induced to stop proliferation, the level of c-myc is found to be downregulated. (International Publication Number WO 91/15580). However, it has been shown that exposure of HL-60 cells to a DNA construct that is complementary to the 5' end of c-myc or c-myb blocks translation of the corresponding mRNAs which downregulates expression of the c-myc or c-myb proteins and causes arrest of cell proliferation and differentiation of the treated cells. (International Publication Number WO 91/15580; Wickstrom et al., Proc. Natl. Acad. Sci. 85:1028 (1988); Anfossi et al., Proc. Natl. Acad. Sci. 86:3379 (1989)). However, the skilled artisan would appreciate the present invention's usefulness is not be limited to treatment, prevention, and/or prognosis of proliferative disorders of cells and tissues of hematopoietic origin, in light of the numerous cells and cell types of varying origins which are known to exhibit proliferative phenotypes.

In addition to the foregoing, a polynucleotide of the present invention can be used to control gene expression through triple helix formation or through antisense DNA or RNA. Antisense techniques are discussed, for example, in Okano, J. Neurochem. 56: 560 (1991); "Oligodeoxynucleotides as Antisense Inhibitors of Gene Expression, CRC Press, Boca Raton, FL (1988). Triple helix formation is discussed in, for instance Lee et al., Nucleic Acids Research 6:

3073 (1979); Cooney et al., Science 241: 456 (1988); and Dervan et al., Science 251: 1360 (1991). Both methods rely on binding of the polynucleotide to a complementary DNA or RNA. For these techniques, preferred polynucleotides are usually oligonucleotides 20 to 40 bases in length and complementary to either the region of the gene involved in transcription (triple helix - see Lee et al., Nucl. Acids Res. 6:3073 (1979); Cooney et al., Science 241:456 (1988); and Dervan et al., Science 251:1360 (1991)) or to the mRNA itself (antisense - Okano, J. Neurochem. 56:560 (1991); Oligodeoxy-nucleotides as Antisense Inhibitors of Gene Expression, CRC Press, Boca Raton, FL (1988)). Triple helix formation optimally results in a shut-off of RNA transcription from DNA, while antisense RNA hybridization blocks translation of an mRNA molecule into polypeptide. The oligonucleotide described above can also be delivered to cells such that the antisense RNA or DNA may be expressed in vivo to inhibit production of polypeptide of the present invention antigens. Both techniques are effective in model systems, and the information disclosed herein can be used to design antisense or triple helix polynucleotides in an effort to treat disease, and in particular, for the treatment of proliferative diseases and/or conditions. Non-limiting antisense and triple helix methods encompassed by the present invention are more thoroughly described elsewhere herein (see, e.g., the section labeled "Antisense and Ribozyme (Antagonists)").

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Polynucleotides of the present invention are also useful in gene therapy. One goal of gene therapy is to insert a normal gene into an organism having a defective gene, in an effort to correct the genetic defect. The polynucleotides disclosed in the present invention offer a means of targeting such genetic defects in a highly accurate manner. Another goal is to insert a new gene that was not present in the host genome, thereby producing a new trait in the host cell. Additional non-limiting examples of gene therapy methods encompassed by the present invention are more thoroughly described elsewhere herein (see, e.g., the sections labeled "Gene Therapy Methods", and Examples 16, 17 and 18).

The polynucleotides are also useful for identifying individuals from minute biological samples. The United States military, for example, is considering the use of restriction fragment length polymorphism (RFLP) for identification of its personnel. In this technique, an individual's genomic DNA is digested with one or more restriction enzymes, and probed on a Southern blot to yield unique bands for identifying personnel. This method does not suffer from the current limitations of "Dog Tags" which can be lost, switched, or stolen, making positive identification difficult. The polynucleotides of the present invention can be used as additional DNA markers for RFLP.

The polynucleotides of the present invention can also be used as an alternative to RFLP, by determining the actual base-by-base DNA sequence of selected portions of an individual's genome. These sequences can be used to prepare PCR primers for amplifying and isolating such selected DNA, which can then be sequenced. Using this technique, individuals can be identified because each individual will have a unique set of DNA sequences. Once a unique ID database is

established for an individual, positive identification of that individual, living or dead, can be made from extremely small tissue samples.

Forensic biology also benefits from using DNA-based identification techniques as disclosed herein. DNA sequences taken from very small biological samples such as tissues, e.g., hair or skin, or body fluids, e.g., blood, saliva, semen, synovial fluid, amniotic fluid, breast milk, lymph, pulmonary sputum or surfactant, urine, fecal matter, etc., can be amplified using PCR. In one prior art technique, gene sequences amplified from polymorphic loci, such as DQa class II HLA gene, are used in forensic biology to identify individuals. (Erlich, H., PCR Technology, Freeman and Co. (1992)). Once these specific polymorphic loci are amplified, they are digested with one or more restriction enzymes, yielding an identifying set of bands on a Southern blot probed with DNA corresponding to the DQa class II HLA gene. Similarly, polynucleotides of the present invention can be used as polymorphic markers for forensic purposes.

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There is also a need for reagents capable of identifying the source of a particular tissue. Such need arises, for example, in forensics when presented with tissue of unknown origin. Appropriate reagents can comprise, for example, DNA probes or primers prepared from the sequences of the present invention, specific to tissues, including but not limited to those shown in Table 1B. Panels of such reagents can identify tissue by species and/or by organ type. In a similar fashion, these reagents can be used to screen tissue cultures for contamination. Additional non-limiting examples of such uses are further described herein.

The polynucleotides of the present invention are also useful as hybridization probes for differential identification of the tissue(s) or cell type(s) present in a biological sample. Similarly, polypeptides and antibodies directed to polypeptides of the present invention are useful to provide immunological probes for differential identification of the tissue(s) (e.g., immunohistochemistry assays) or cell type(s) (e.g., immunocytochemistry assays). In addition, for a number of disorders of the above tissues or cells, significantly higher or lower levels of gene expression of the polynucleotides/polypeptides of the present invention may be detected in certain tissues (e.g., tissues expressing polypeptides and/or polynucleotides of the present invention, for example, those disclosed in column 5 of Table 1B.2, and/or cancerous and/or wounded tissues) or bodily fluids (e.g., semen, lymph, vaginal pool, serum, plasma, urine, synovial fluid or spinal fluid) taken from an individual having such a disorder, relative to a "standard" gene expression level, i.e., the expression level in healthy tissue from an individual not having the disorder.

Thus, the invention provides a diagnostic method of a disorder, which involves: (a) assaying gene expression level in cells or body fluid of an individual; (b) comparing the gene expression level with a standard gene expression level, whereby an increase or decrease in the assayed gene expression level compared to the standard expression level is indicative of a disorder.

In the very least, the polynucleotides of the present invention can be used as molecular weight markers on Southern gels, as diagnostic probes for the presence of a specific mRNA in a particular cell type, as a probe to "subtract-out" known sequences in the process of discovering novel polynucleotides, for selecting and making oligomers for attachment to a "gene chip" or other support, to raise anti-DNA antibodies using DNA immunization techniques, and as an antigen to elicit an immune response.

# Uses of the Polypeptides

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Each of the polypeptides identified herein can be used in numerous ways. The following description should be considered exemplary and utilizes known techniques.

Polypeptides and antibodies directed to polypeptides of the present invention are useful to provide immunological probes for differential identification of the tissue(s) (e.g., immunohistochemistry assays such as, for example, ABC immunoperoxidase (Hsu et al., J. Histochem. Cytochem. 29:577-580 (1981)) or cell type(s) (e.g., immunocytochemistry assays).

Antibodies can be used to assay levels of polypeptides encoded by polynucleotides of the invention in a biological sample using classical immunohistological methods known to those of skill in the art (e.g., see Jalkanen, et al., J. Cell. Biol. 101:976-985 (1985); Jalkanen, et al., J. Cell. Biol. 105:3087-3096 (1987)). Other antibody-based methods useful for detecting protein gene expression include immunoassays, such as the enzyme linked immunosorbent assay (ELISA) and the radioimmunoassay (RIA). Suitable antibody assay labels are known in the art and include enzyme labels, such as, glucose oxidase; radioisotopes, such as iodine (131 I, 125 I, 123 I, 121 I), carbon (14C), sulfur (35S), tritium (3H), indium (115mIn, 113mIn, 112In, 111 In), and technetium (99Tc, 99mTc), thallium (201Ti), gallium (68Ga, 67Ga), palladium (103Pd), molybdenum (99Mo), xenon (133Xe), fluorine (18F), 153Sm, 177Lu, 159Gd, 149Pm, 140La, 175Yb, 166Ho, 90Y, 47Sc, 186Re, 188Re, 142Pr, 105Rh, 97Ru; luminescent labels, such as luminol; and fluorescent labels, such as fluorescein and rhodamine, and biotin.

In addition to assaying levels of polypeptide of the present invention in a biological sample, proteins can also be detected *in vivo* by imaging. Antibody labels or markers for *in vivo* imaging of protein include those detectable by X-radiography, NMR or ESR. For X-radiography, suitable labels include radioisotopes such as barium or cesium, which emit detectable radiation but are not overtly harmful to the subject. Suitable markers for NMR and ESR include those with a detectable characteristic spin, such as deuterium, which may be incorporated into the antibody by labeling of nutrients for the relevant hybridoma.

A protein-specific antibody or antibody fragment which has been labeled with an appropriate detectable imaging moiety, such as a radioisotope (for example, ¹³¹I, ¹¹²In, ^{99m}Tc, (¹³¹I, ¹²⁵I, ¹²³I, ¹²¹I), carbon (¹⁴C), sulfur (³⁵S), tritium (³H), indium (^{115m}In, ^{113m}In, ¹¹²In, ¹¹¹In), and technetium (⁹⁹Tc, ^{99m}Tc), thallium (²⁰¹Ti), gallium (⁶⁸Ga, ⁶⁷Ga), palladium (¹⁰³Pd), molybdenum

(99Mo), xenon (133Xe), fluorine (18F, 153Sm, 177Lu, 159Gd, 149Pm, 140La, 175Yb, 166Ho, 90Y, 47Sc, 186Re, 188Re, 142Pr, 105Rh, 97Ru), a radio-opaque substance, or a material detectable by nuclear magnetic resonance, is introduced (for example, parenterally, subcutaneously or intraperitoneally) into the mammal to be examined for immune system disorder. It will be understood in the art that the size of the subject and the imaging system used will determine the quantity of imaging moiety needed to produce diagnostic images. In the case of a radioisotope moiety, for a human subject, the quantity of radioactivity injected will normally range from about 5 to 20 millicuries of 99mTc. The labeled antibody or antibody fragment will then preferentially accumulate at the location of cells which express the polypeptide encoded by a polynucleotide of the invention. *In vivo* tumor imaging is described in S.W. Burchiel et al., "Immunopharmacokinetics of Radiolabeled Antibodies and Their Fragments" (Chapter 13 in *Tumor Imaging: The Radiochemical Detection of Cancer*, S.W. Burchiel and B. A. Rhodes, eds., Masson Publishing Inc. (1982)).

In one embodiment, the invention provides a method for the specific delivery of compositions of the invention to cells by administering polypeptides of the invention (e.g., polypeptides encoded by polynucleotides of the invention and/or antibodies) that are associated with heterologous polypeptides or nucleic acids. In one example, the invention provides a method for delivering a therapeutic protein into the targeted cell. In another example, the invention provides a method for delivering a single stranded nucleic acid (e.g., antisense or ribozymes) or double stranded nucleic acid (e.g., DNA that can integrate into the cell's genome or replicate episomally and that can be transcribed) into the targeted cell.

In another embodiment, the invention provides a method for the specific destruction of cells (e.g., the destruction of tumor cells) by administering polypeptides of the invention in association with toxins or cytotoxic prodrugs.

By "toxin" is meant one or more compounds that bind and activate endogenous cytotoxic effector systems, radioisotopes, holotoxins, modified toxins, catalytic subunits of toxins, or any molecules or enzymes not normally present in or on the surface of a cell that under defined conditions cause the cell's death. Toxins that may be used according to the methods of the invention include, but are not limited to, radioisotopes known in the art, compounds such as, for example, antibodies (or complement fixing containing portions thereof) that bind an inherent or induced endogenous cytotoxic effector system, thymidine kinase, endonuclease, RNAse, alpha toxin, ricin, abrin, *Pseudomonas* exotoxin A, diphtheria toxin, saporin, momordin, gelonin, pokeweed antiviral protein, alpha-sarcin and cholera toxin. "Toxin" also includes a cytostatic or cytocidal agent, a therapeutic agent or a radioactive metal ion, e.g., alpha-emitters such as, for example, ²¹³Bi, or other radioisotopes such as, for example, ¹⁰³Pd, ¹³³Xe, ¹³¹I, ⁶⁸Ge, ⁵⁷Co, ⁶⁵Zn, ⁸⁵Sr, ³²P, ³⁵S, ⁹⁰Y, ¹⁵³Sm, ¹⁵³Gd, ¹⁶⁹Yb, ⁵¹Cr, ⁵⁴Mn, ⁷⁵Se, ¹¹³Sn, ⁹⁰Yttrium, ¹¹⁷Tin, ¹⁸⁶Rhenium, ¹⁶⁶Holmium, and ¹⁸⁸Rhenium; luminescent labels, such as luminol; and fluorescent labels, such as fluorescein and rhodamine, and biotin. In a specific embodiment, the invention provides a method

for the specific destruction of cells (e.g., the destruction of tumor cells) by administering polypeptides of the invention or antibodies of the invention in association with the radioisotope ⁹⁰Y. In another specific embodiment, the invention provides a method for the specific destruction of cells (e.g., the destruction of tumor cells) by administering polypeptides of the invention or antibodies of the invention in association with the radioisotope ¹¹¹In. In a further specific embodiment, the invention provides a method for the specific destruction of cells (e.g., the destruction of tumor cells) by administering polypeptides of the invention or antibodies of the invention in association with the radioisotope ¹³¹I.

Techniques known in the art may be applied to label polypeptides of the invention (including antibodies). Such techniques include, but are not limited to, the use of bifunctional conjugating agents (see e.g., U.S. Patent Nos. 5,756,065; 5,714,631; 5,696,239; 5,652,361; 5,505,931; 5,489,425; 5,435,990; 5,428,139; 5,342,604; 5,274,119; 4,994,560; and 5,808,003; the contents of each of which are hereby incorporated by reference in its entirety).

Thus, the invention provides a diagnostic method of a disorder, which involves (a) assaying the expression level of a polypeptide of the present invention in cells or body fluid of an individual; and (b) comparing the assayed polypeptide expression level with a standard polypeptide expression level, whereby an increase or decrease in the assayed polypeptide expression level compared to the standard expression level is indicative of a disorder. With respect to cancer, the presence of a relatively high amount of transcript in biopsied tissue from an individual may indicate a predisposition for the development of the disease, or may provide a means for detecting the disease prior to the appearance of actual clinical symptoms. A more definitive diagnosis of this type may allow health professionals to employ preventative measures or aggressive treatment earlier thereby preventing the development or further progression of the cancer.

Moreover, polypeptides of the present invention can be used to treat or prevent diseases or conditions such as, for example, neural disorders, immune system disorders, muscular disorders, reproductive disorders, gastrointestinal disorders, pulmonary disorders, cardiovascular disorders, renal disorders, proliferative disorders, and/or cancerous diseases and conditions. For example, patients can be administered a polypeptide of the present invention in an effort to replace absent or decreased levels of the polypeptide (e.g., insulin), to supplement absent or decreased levels of a different polypeptide (e.g., hemoglobin S for hemoglobin B, SOD, catalase, DNA repair proteins), to inhibit the activity of a polypeptide (e.g., an oncogene or tumor supressor), to activate the activity of a polypeptide (e.g., by binding to a receptor), to reduce the activity of a membrane bound receptor by competing with it for free ligand (e.g., soluble TNF receptors used in reducing inflammation), or to bring about a desired response (e.g., blood vessel growth inhibition, enhancement of the immune response to proliferative cells or tissues).

Similarly, antibodies directed to a polypeptide of the present invention can also be used to treat disease (as described *supra*, and elsewhere herein). For example, administration of an antibody directed to a polypeptide of the present invention can bind, and/or neutralize the polypeptide, and/or reduce overproduction of the polypeptide. Similarly, administration of an antibody can activate the polypeptide, such as by binding to a polypeptide bound to a membrane (receptor).

At the very least, the polypeptides of the present invention can be used as molecular weight markers on SDS-PAGE gels or on molecular sieve gel filtration columns using methods well known to those of skill in the art. Polypeptides can also be used to raise antibodies, which in turn are used to measure protein expression from a recombinant cell, as a way of assessing transformation of the host cell. Moreover, the polypeptides of the present invention can be used to test the biological activities described herein.

### Diagnostic Assays

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The compounds of the present invention are useful for diagnosis, treatment, prevention and/or prognosis of various disorders in mammals, preferably humans. Such disorders include, but are not limited to, those related to biological activities described in Table 1D and, also as described herein under the section heading "Biological Activities".

For a number of disorders, substantially altered (increased or decreased) levels of gene expression can be detected in tissues, cells or bodily fluids (e.g., sera, plasma, urine, semen, synovial fluid or spinal fluid) taken from an individual having such a disorder, relative to a "standard" gene expression level, that is, the expression level in tissues or bodily fluids from an individual not having the disorder. Thus, the invention provides a diagnostic method useful during diagnosis of a disorder, which involves measuring the expression level of the gene encoding the polypeptide in tissues, cells or body fluid from an individual and comparing the measured gene expression level with a standard gene expression level, whereby an increase or decrease in the gene expression level(s) compared to the standard is indicative of a disorder. These diagnostic assays may be performed *in vivo* or *in vitro*, such as, for example, on blood samples, biopsy tissue or autopsy tissue.

The present invention is also useful as a prognostic indicator, whereby patients exhibiting enhanced or depressed gene expression will experience a worse clinical outcome relative to patients expressing the gene at a level nearer the standard level.

In certain embodiments, a polypeptide of the invention, or polynucleotides, antibodies, agonists, or antagonists corresponding to that polypeptide, may be used to diagnose and/or prognosticate diseases and/or disorders associated with the tissue(s) in which the polypeptide of the invention is expressed, including one, two, three, four, five, or more tissues disclosed in Table 1B.2, column 5 (Tissue Distribution Library Code).

By "assaying the expression level of the gene encoding the polypeptide" is intended qualitatively or quantitatively measuring or estimating the level of the polypeptide of the invention or the level of the mRNA encoding the polypeptide of the invention in a first biological sample either directly (e.g., by determining or estimating absolute protein level or mRNA level) or relatively (e.g., by comparing to the polypeptide level or mRNA level in a second biological sample). Preferably, the polypeptide expression level or mRNA level in the first biological sample is measured or estimated and compared to a standard polypeptide level or mRNA level, the standard being taken from a second biological sample obtained from an individual not having the disorder or being determined by averaging levels from a population of individuals not having the disorder. As will be appreciated in the art, once a standard polypeptide level or mRNA level is known, it can be used repeatedly as a standard for comparison.

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By "biological sample" is intended any biological sample obtained from an individual, cell line, tissue culture, or other source containing polypeptides of the invention (including portions thereof) or mRNA. As indicated, biological samples include body fluids (such as sera, plasma, urine, synovial fluid and spinal fluid) and tissue sources found to express the full length or fragments thereof of a polypeptide or mRNA. Methods for obtaining tissue biopsies and body fluids from mammals are well known in the art. Where the biological sample is to include mRNA, a tissue biopsy is the preferred source.

Total cellular RNA can be isolated from a biological sample using any suitable technique such as the single-step guanidinium-thiocyanate-phenol-chloroform method described in Chomczynski and Sacchi, Anal. Biochem. 162:156-159 (1987). Levels of mRNA encoding the polypeptides of the invention are then assayed using any appropriate method. These include Northern blot analysis, S1 nuclease mapping, the polymerase chain reaction (PCR), reverse transcription in combination with the polymerase chain reaction (RT-PCR), and reverse transcription in combination with the ligase chain reaction (RT-LCR).

The present invention also relates to diagnostic assays such as quantitative and diagnostic assays for detecting levels of polypeptides of the invention, in a biological sample (e.g., cells and tissues), including determination of normal and abnormal levels of polypeptides. Thus, for instance, a diagnostic assay in accordance with the invention for detecting over-expression of polypeptides of the invention compared to normal control tissue samples may be used to detect the presence of tumors. Assay techniques that can be used to determine levels of a polypeptide, such as a polypeptide of the present invention in a sample derived from a host are well-known to those of skill in the art. Such assay methods include radioimmunoassays, competitive-binding assays, Western Blot analysis and ELISA assays. Assaying polypeptide levels in a biological sample can occur using any art-known method.

Assaying polypeptide levels in a biological sample can occur using antibody-based techniques. For example, polypeptide expression in tissues can be studied with classical

immunohistological methods (Jalkanen et al., J. Cell. Biol. 101:976-985 (1985); Jalkanen, M., et al., J. Cell. Biol. 105:3087-3096 (1987)). Other antibody-based methods useful for detecting polypeptide gene expression include immunoassays, such as the enzyme linked immunosorbent assay (ELISA) and the radioimmunoassay (RIA). Suitable antibody assay labels are known in the art and include enzyme labels, such as, glucose oxidase, and radioisotopes, such as iodine (125 I, 121 I), carbon (14 C), sulfur (35 S), tritium (3H), indium (112 In), and technetium (99 Tc), and fluorescent labels, such as fluorescein and rhodamine, and biotin.

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The tissue or cell type to be analyzed will generally include those which are known, or suspected, to express the gene of inteest (such as, for example, cancer). The protein isolation methods employed herein may, for example, be such as those described in Harlow and Lane (Harlow, E. and Lane, D., 1988, "Antibodies: A Laboratory Manual", Cold Spring Harbor Laboratory Press, Cold Spring Harbor, New York), which is incorporated herein by reference in its entirety. The isolated cells can be derived from cell culture or from a patient. The analysis of cells taken from culture may be a necessary step in the assessment of cells that could be used as part of a cell-based gene therapy technique or, alternatively, to test the effect of compounds on the expression of the gene.

For example, antibodies, or fragments of antibodies, such as those described herein, may be used to quantitatively or qualitatively detect the presence of gene products or conserved variants or peptide fragments thereof. This can be accomplished, for example, by immunofluorescence techniques employing a fluorescently labeled antibody coupled with light microscopic, flow cytometric, or fluorimetric detection.

In a preferred embodiment, antibodies, or fragments of antibodies directed to any one or all of the predicted epitope domains of the polypeptides of the invention (shown in Table 1B) may be used to quantitatively or qualitatively detect the presence of gene products or conserved variants or peptide fragments thereof. This can be accomplished, for example, by immunofluorescence techniques employing a fluorescently labeled antibody coupled with light microscopic, flow cytometric, or fluorimetric detection.

In an additional preferred embodiment, antibodies, or fragments of antibodies directed to a conformational epitope of a polypeptide of the invention may be used to quantitatively or qualitatively detect the presence of gene products or conserved variants or peptide fragments thereof. This can be accomplished, for example, by immunofluorescence techniques employing a fluorescently labeled antibody coupled with light microscopic, flow cytometric, or fluorimetric detection.

The antibodies (or fragments thereof), and/or polypeptides of the present invention may, additionally, be employed histologically, as in immunofluorescence, immunoelectron microscopy or non-immunological assays, for in situ detection of gene products or conserved variants or peptide fragments thereof. In situ detection may be accomplished by removing a histological

specimen from a patient, and applying thereto a labeled antibody or polypeptide of the present invention. The antibody (or fragment thereof) or polypeptide is preferably applied by overlaying the labeled antibody (or fragment) onto a biological sample. Through the use of such a procedure, it is possible to determine not only the presence of the gene product, or conserved variants or peptide fragments, or polypeptide binding, but also its distribution in the examined tissue. Using the present invention, those of ordinary skill will readily perceive that any of a wide variety of histological methods (such as staining procedures) can be modified in order to achieve such in situ detection.

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Immunoassays and non-immunoassays for gene products or conserved variants or peptide fragments thereof will typically comprise incubating a sample, such as a biological fluid, a tissue extract, freshly harvested cells, or lysates of cells which have been incubated in cell culture, in the presence of a detectably labeled antibody capable of binding gene products or conserved variants or peptide fragments thereof, and detecting the bound antibody by any of a number of techniques well-known in the art.

The biological sample may be brought in contact with and immobilized onto a solid phase support or carrier such as nitrocellulose, or other solid support which is capable of immobilizing cells, cell particles or soluble proteins. The support may then be washed with suitable buffers followed by treatment with the detectably labeled antibody or detectable polypeptide of the invention. The solid phase support may then be washed with the buffer a second time to remove unbound antibody or polypeptide. Optionally the antibody is subsequently labeled. The amount of bound label on solid support may then be detected by conventional means.

By "solid phase support or carrier" is intended any support capable of binding an antigen or an antibody. Well-known supports or carriers include glass, polystyrene, polypropylene, polyethylene, dextran, nylon, amylases, natural and modified celluloses, polyacrylamides, gabbros, and magnetite. The nature of the carrier can be either soluble to some extent or insoluble for the purposes of the present invention. The support material may have virtually any possible structural configuration so long as the coupled molecule is capable of binding to an antigen or antibody. Thus, the support configuration may be spherical, as in a bead, or cylindrical, as in the inside surface of a test tube, or the external surface of a rod. Alternatively, the surface may be flat such as a sheet, test strip, etc. Preferred supports include polystyrene beads. Those skilled in the art will know many other suitable carriers for binding antibody or antigen, or will be able to ascertain the same by use of routine experimentation.

The binding activity of a given lot of antibody or antigen polypeptide may be determined according to well known methods. Those skilled in the art will be able to determine operative and optimal assay conditions for each determination by employing routine experimentation.

In addition to assaying polypeptide levels or polynucleotide levels in a biological sample obtained from an individual, polypeptide or polynucleotide can also be detected *in vivo* by

imaging. For example, in one embodiment of the invention, polypeptides and/or antibodies of the invention are used to image diseased cells, such as neoplasms. In another embodiment, polynucleotides of the invention (e.g., polynucleotides complementary to all or a portion of an mRNA) and/or antibodies (e.g., antibodies directed to any one or a combination of the epitopes of a polypeptide of the invention, antibodies directed to a conformational epitope of a polypeptide of the invention, or antibodies directed to the full length polypeptide expressed on the cell surface of a mammalian cell) are used to image diseased or neoplastic cells.

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Antibody labels or markers for *in vivo* imaging of polypeptides of the invention include those detectable by X-radiography, NMR, MRI, CAT-scans or ESR. For X-radiography, suitable labels include radioisotopes such as barium or cesium, which emit detectable radiation but are not overtly harmful to the subject. Suitable markers for NMR and ESR include those with a detectable characteristic spin, such as deuterium, which may be incorporated into the antibody by labeling of nutrients for the relevant hybridoma. Where *in vivo* imaging is used to detect enhanced levels of polypeptides for diagnosis in humans, it may be preferable to use human antibodies or "humanized" chimeric monoclonal antibodies. Such antibodies can be produced using techniques described herein or otherwise known in the art. For example methods for producing chimeric antibodies are known in the art. See, for review, Morrison, *Science* 229:1202 (1985); Oi et al., *BioTechniques* 4:214 (1986); Cabilly et al., U.S. Patent No. 4,816,567; Taniguchi et al., EP 171496; Morrison et al., EP 173494; Neuberger et al., WO 8601533; Robinson et al., WO 8702671; Boulianne et al., *Nature* 312:643 (1984); Neuberger et al., *Nature* 314:268 (1985).

Additionally, any polypeptides of the invention whose presence can be detected, can be administered. For example, polypeptides of the invention labeled with a radio-opaque or other appropriate compound can be administered and visualized *in vivo*, as discussed, above for labeled antibodies. Further, such polypeptides can be utilized for *in vitro* diagnostic procedures.

A polypeptide-specific antibody or antibody fragment which has been labeled with an appropriate detectable imaging moiety, such as a radioisotope (for example, ¹³¹I, ¹¹²In, ^{99m}Tc), a radio-opaque substance, or a material detectable by nuclear magnetic resonance, is introduced (for example, parenterally, subcutaneously or intraperitoneally) into the mammal to be examined for a disorder. It will be understood in the art that the size of the subject and the imaging system used will determine the quantity of imaging moiety needed to produce diagnostic images. In the case of a radioisotope moiety, for a human subject, the quantity of radioactivity injected will normally range from about 5 to 20 millicuries of ^{99m}Tc. The labeled antibody or antibody fragment will then preferentially accumulate at the location of cells which contain the antigenic protein. *In vivo* tumor imaging is described in S.W. Burchiel et al., "Immunopharmacokinetics of Radiolabeled Antibodies and Their Fragments" (Chapter 13 in *Tumor Imaging: The Radiochemical Detection of Cancer*, S.W. Burchiel and B. A. Rhodes, eds., Masson Publishing Inc. (1982)).

With respect to antibodies, one of the ways in which an antibody of the present invention

can be detectably labeled is by linking the same to a reporter enzyme and using the linked product in an enzyme immunoassay (EIA) (Voller, A., "The Enzyme Linked Immunosorbent Assay (ELISA)", 1978, Diagnostic Horizons 2:1-7, Microbiological Associates Quarterly Publication, Walkersville, MD); Voller et al., J. Clin. Pathol. 31:507-520 (1978); Butler, J.E., Meth. Enzymol. 73:482-523 (1981); Maggio, E. (ed.), 1980, Enzyme Immunoassay, CRC Press, Boca Raton, FL; Ishikawa, E. et al., (eds.), 1981, Enzyme Immunoassay, Kgaku Shoin, Tokyo). The reporter enzyme which is bound to the antibody will react with an appropriate substrate, preferably a chromogenic substrate, in such a manner as to produce a chemical moiety which can be detected, for example, by spectrophotometric, fluorimetric or by visual means. Reporter enzymes which can be used to detectably label the antibody include, but are not limited to, malate dehydrogenase, staphylococcal nuclease, delta-5-steroid isomerase, yeast alcohol dehydrogenase, alphaglycerophosphate, dehydrogenase, triose phosphate isomerase, horseradish peroxidase, alkaline phosphatase, asparaginase, glucose oxidase, beta-galactosidase, ribonuclease, urease, catalase, glucose-6-phosphate dehydrogenase, glucoamylase and acetylcholinesterase. Additionally, the detection can be accomplished by colorimetric methods which employ a chromogenic substrate for the reporter enzyme. Detection may also be accomplished by visual comparison of the extent of enzymatic reaction of a substrate in comparison with similarly prepared standards.

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Detection may also be accomplished using any of a variety of other immunoassays. For example, by radioactively labeling the antibodies or antibody fragments, it is possible to detect polypeptides through the use of a radioimmunoassay (RIA) (see, for example, Weintraub, B., Principles of Radioimmunoassays, Seventh Training Course on Radioligand Assay Techniques, The Endocrine Society, March, 1986, which is incorporated by reference herein). The radioactive isotope can be detected by means including, but not limited to, a gamma counter, a scintillation counter, or autoradiography.

It is also possible to label the antibody with a fluorescent compound. When the fluorescently labeled antibody is exposed to light of the proper wave length, its presence can then be detected due to fluorescence. Among the most commonly used fluorescent labeling compounds are fluorescein isothiocyanate, rhodamine, phycocyanin, phycocyanin, allophycocyanin, ophthaldehyde and fluorescamine.

The antibody can also be detectably labeled using fluorescence emitting metals such as ¹⁵²Eu, or others of the lanthanide series. These metals can be attached to the antibody using such metal chelating groups as diethylenetriaminepentacetic acid (DTPA) or ethylenediaminetetraacetic acid (EDTA).

The antibody also can be detectably labeled by coupling it to a chemiluminescent compound. The presence of the chemiluminescent-tagged antibody is then determined by detecting the presence of luminescence that arises during the course of a chemical reaction. Examples of particularly useful chemiluminescent labeling compounds are luminol, isoluminol,

theromatic acridinium ester, imidazole, acridinium salt and oxalate ester.

Likewise, a bioluminescent compound may be used to label the antibody of the present invention. Bioluminescence is a type of chemiluminescence found in biological systems in, which a catalytic protein increases the efficiency of the chemiluminescent reaction. The presence of a bioluminescent protein is determined by detecting the presence of luminescence. Important bioluminescent compounds for purposes of labeling are luciferin, luciferase and aequorin.

#### Methods for Detecting Diseases

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In general, a disease may be detected in a patient based on the presence of one or more proteins of the invention and/or polynucleotides encoding such proteins in a biological sample (for example, blood, sera, urine, and/or tumor biopsies) obtained from the patient. In other words, such proteins may be used as markers to indicate the presence or absence of a disease or disorder, including cancer and/or as described elsewhere herein. In addition, such proteins may be useful for the detection of other diseases and cancers. The binding agents provided herein generally permit detection of the level of antigen that binds to the agent in the biological sample. Polynucleotide primers and probes may be used to detect the level of mRNA encoding polypeptides of the invention, which is also indicative of the presence or absence of a disease or disorder, including cancer. In general, polypeptides of the invention should be present at a level that is at least three fold higher in diseased tissue than in normal tissue.

There are a variety of assay formats known to those of ordinary skill in the art for using a binding agent to detect polypeptide markers in a sample. See, e.g., Harlow and Lane, *supra*. In general, the presence or absence of a disease in a patient may be determined by (a) contacting a biological sample obtained from a patient with a binding agent; (b) detecting in the sample a level of polypeptide that binds to the binding agent; and (c) comparing the level of polypeptide with a predetermined cut-off value.

In a preferred embodiment, the assay involves the use of a binding agent(s) immobilized on a solid support to bind to and remove the polypeptide of the invention from the remainder of the sample. The bound polypeptide may then be detected using a detection reagent that contains a reporter group and specifically binds to the binding agent/polypeptide complex. Such detection reagents may comprise, for example, a binding agent that specifically binds to the polypeptide or an antibody or other agent that specifically binds to the binding agent, such as an anti-immunoglobulin, protein G, protein A or a lectin. Alternatively, a competitive assay may be utilized, in which a polypeptide is labeled with a reporter group and allowed to bind to the immobilized binding agent after incubation of the binding agent with the sample. The extent to which components of the sample inhibit the binding of the labeled polypeptide to the binding agent is indicative of the reactivity of the sample with the immobilized binding agent. Suitable

polypeptides for use within such assays include polypeptides of the invention and portions thereof, or antibodies, to which the binding agent binds, as described above.

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The solid support may be any material known to those of skill in the art to which polypeptides of the invention may be attached. For example, the solid support may be a test well in a microtiter plate or a nitrocellulose or other suitable membrane. Alternatively, the support may be a bead or disc, such as glass fiberglass, latex or a plastic material such as polystyrene or polyvinylchloride. The support may also be a magnetic particle or a fiber optic sensor, such as those disclosed, for example, in U.S. Patent No. 5,359,681. The binding agent may be immobilized on the solid support using a variety of techniques known to those of skill in the art, which are amply described in the patent and scientific literature. In the context of the present invention, the term "immobilization" refers to both noncovalent association, such as adsorption, and covalent attachment (which may be a direct linkage between the agent and functional groups on the support or may be a linkage by way of a cross-linking agent). Immobilization by adsorption to a well in a microtiter plate or to a membrane is preferred. In such cases, adsorption may be achieved by contacting the binding agent, in a suitable buffer, with the solid support for the suitable amount of time. The contact time varies with temperature, but is typically between about 1 hour and about 1 day. In general, contacting a well of plastic microtiter plate (such as polystyrene or polyvinylchloride) with an amount of binding agent ranging from about 10 ng to about 10 ug, and preferably about 100 ng to about 1 ug, is sufficient to immobilize an adequate amount of binding agent.

Covalent attachment of binding agent to a solid support may generally be achieved by first reacting the support with a bifunctional reagent that will react with both the support and a functional group, such as a hydroxyl or amino group, on the binding agent. For example, the binding agent may be covalently attached to supports having an appropriate polymer coating using benzoquinone or by condensation of an aldehyde group on the support with an amine and an active hydrogen on the binding partner (see, e.g., Pierce Immunotechnology Catalog and Handbook, 1991, at A12-A13).

# Gene Therapy Methods

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Also encompassed by the invention are gene therapy methods for treating or preventing disorders, diseases and conditions. The gene therapy methods relate to the introduction of nucleic acid (DNA, RNA and antisense DNA or RNA) sequences into an animal to achieve expression of the polypeptide of the present invention. This method requires a polynucleotide which codes for a polypeptide of the present invention operatively linked to a promoter and any other genetic elements necessary for the expression of the polypeptide by the target tissue. Such gene therapy and delivery techniques are known in the art, see, for example, WO90/11092, which is herein incorporated by reference.

Thus, for example, cells from a patient may be engineered with a polynucleotide (DNA or RNA) comprising a promoter operably linked to a polynucleotide of the present invention ex vivo, with the engineered cells then being provided to a patient to be treated with the polypeptide of the present invention. Such methods are well-known in the art. For example, see Belldegrun, A., et al., J. Natl. Cancer Inst. 85: 207-216 (1993); Ferrantini, M. et al., Cancer Research 53: 1107-1112 (1993); Ferrantini, M. et al., J. Immunology 153: 4604-4615 (1994); Kaido, T., et al., Int. J. Cancer 60: 221-229 (1995); Ogura, H., et al., Cancer Research 50: 5102-5106 (1990); Santodonato, L., et al., Human Gene Therapy 7:1-10 (1996); Santodonato, L., et al., Gene Therapy 4:1246-1255 (1997); and Zhang, J.-F. et al., Cancer Gene Therapy 3: 31-38 (1996)), which are herein incorporated by reference. In one embodiment, the cells which are engineered are arterial cells. The arterial cells may be reintroduced into the patient through direct injection to the artery, the tissues surrounding the artery, or through catheter injection.

As discussed in more detail below, the polynucleotide constructs can be delivered by any method that delivers injectable materials to the cells of an animal, such as, injection into the interstitial space of tissues (heart, muscle, skin, lung, liver, and the like). The polynucleotide constructs may be delivered in a pharmaceutically acceptable liquid or aqueous carrier.

In one embodiment, the polynucleotide of the present invention is delivered as a naked polynucleotide. The term "naked" polynucleotide, DNA or RNA refers to sequences that are free from any delivery vehicle that acts to assist, promote or facilitate entry into the cell, including viral sequences, viral particles, liposome formulations, lipofectin or precipitating agents and the like. However, the polynucleotide of the present invention can also be delivered in liposome formulations and lipofectin formulations and the like can be prepared by methods well known to those skilled in the art. Such methods are described, for example, in U.S. Patent Nos. 5,593,972, 5,589,466, and 5,580,859, which are herein incorporated by reference.

The polynucleotide vector constructs used in the gene therapy method are preferably constructs that will not integrate into the host genome nor will they contain sequences that allow for replication. Appropriate vectors include pWLNEO, pSV2CAT, pOG44, pXT1 and pSG available from Stratagene; pSVK3, pBPV, pMSG and pSVL available from Pharmacia; and

pEF1/V5, pcDNA3.1, and pRc/CMV2 available from Invitrogen. Other suitable vectors will be readily apparent to the skilled artisan.

Any strong promoter known to those skilled in the art can be used for driving the expression of the polynucleotide sequence. Suitable promoters include adenoviral promoters, such as the adenoviral major late promoter; or heterologous promoters, such as the cytomegalovirus (CMV) promoter; the respiratory syncytial virus (RSV) promoter; inducible promoters, such as the MMT promoter, the metallothionein promoter; heat shock promoters; the albumin promoter; the ApoAI promoter; human globin promoters; viral thymidine kinase promoters, such as the Herpes Simplex thymidine kinase promoter; retroviral LTRs; the b-actin promoter; and human growth hormone promoters. The promoter also may be the native promoter for the polynucleotide of the present invention.

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Unlike other gene therapy techniques, one major advantage of introducing naked nucleic acid sequences into target cells is the transitory nature of the polynucleotide synthesis in the cells. Studies have shown that non-replicating DNA sequences can be introduced into cells to provide production of the desired polypeptide for periods of up to six months.

The polynucleotide construct can be delivered to the interstitial space of tissues within the an animal, including of muscle, skin, brain, lung, liver, spleen, bone marrow, thymus, heart, lymph, blood, bone, cartilage, pancreas, kidney, gall bladder, stomach, intestine, testis, ovary, uterus, rectum, nervous system, eye, gland, and connective tissue. Interstitial space of the tissues comprises the intercellular, fluid, mucopolysaccharide matrix among the reticular fibers of organ tissues, elastic fibers in the walls of vessels or chambers, collagen fibers of fibrous tissues, or that same matrix within connective tissue ensheathing muscle cells or in the lacunae of bone. It is similarly the space occupied by the plasma of the circulation and the lymph fluid of the lymphatic channels. Delivery to the interstitial space of muscle tissue is preferred for the reasons discussed below. They may be conveniently delivered by injection into the tissues comprising these cells. They are preferably delivered to and expressed in persistent, non-dividing cells which are differentiated, although delivery and expression may be achieved in non-differentiated or less completely differentiated cells, such as, for example, stem cells of blood or skin fibroblasts. *In vivo* muscle cells are particularly competent in their ability to take up and express polynucleotides.

For the naked nucleic acid sequence injection, an effective dosage amount of DNA or RNA will be in the range of from about 0.05 mg/kg body weight to about 50 mg/kg body weight. Preferably the dosage will be from about 0.005 mg/kg to about 20 mg/kg and more preferably from about 0.05 mg/kg to about 5 mg/kg. Of course, as the artisan of ordinary skill will appreciate, this dosage will vary according to the tissue site of injection. The appropriate and effective dosage of nucleic acid sequence can readily be determined by those of ordinary skill in the art and may depend on the condition being treated and the route of administration.

The preferred route of administration is by the parenteral route of injection into the interstitial space of tissues. However, other parenteral routes may also be used, such as, inhalation of an aerosol formulation particularly for delivery to lungs or bronchial tissues, throat or mucous membranes of the nose. In addition, naked DNA constructs can be delivered to arteries during angioplasty by the catheter used in the procedure.

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The naked polynucleotides are delivered by any method known in the art, including, but not limited to, direct needle injection at the delivery site, intravenous injection, topical administration, catheter infusion, and so-called "gene guns". These delivery methods are known in the art.

The constructs may also be delivered with delivery vehicles such as viral sequences, viral particles, liposome formulations, lipofectin, precipitating agents, etc. Such methods of delivery are known in the art.

In certain embodiments, the polynucleotide constructs are complexed in a liposome preparation. Liposomal preparations for use in the instant invention include cationic (positively charged), anionic (negatively charged) and neutral preparations. However, cationic liposomes are particularly preferred because a tight charge complex can be formed between the cationic liposome and the polyanionic nucleic acid. Cationic liposomes have been shown to mediate intracellular delivery of plasmid DNA (Felgner et al., Proc. Natl. Acad. Sci. USA (1987) 84:7413-7416, which is herein incorporated by reference); mRNA (Malone et al., Proc. Natl. Acad. Sci. USA (1989) 86:6077-6081, which is herein incorporated by reference); and purified transcription factors (Debs et al., J. Biol. Chem. (1990) 265:10189-10192, which is herein incorporated by reference), in functional form.

Cationic liposomes are readily available. For example, N[1-2,3-dioleyloxy)propyl]-N,N,N-triethylammonium (DOTMA) liposomes are particularly useful and are available under the trademark Lipofectin, from GIBCO BRL, Grand Island, N.Y. (See, also, Felgner et al., Proc. Natl Acad. Sci. USA (1987) 84:7413-7416, which is herein incorporated by reference). Other commercially available liposomes include transfectace (DDAB/DOPE) and DOTAP/DOPE (Boehringer).

Other cationic liposomes can be prepared from readily available materials using techniques well known in the art. See, e.g. PCT Publication No. WO 90/11092 (which is herein incorporated by reference) for a description of the synthesis of DOTAP (1,2-bis(oleoyloxy)-3-(trimethylammonio)propane) liposomes. Preparation of DOTMA liposomes is explained in the literature, see, e.g., P. Felgner et al., Proc. Natl. Acad. Sci. USA 84:7413-7417, which is herein incorporated by reference. Similar methods can be used to prepare liposomes from other cationic lipid materials.

Similarly, anionic and neutral liposomes are readily available, such as from Avanti Polar Lipids (Birmingham, Ala.), or can be easily prepared using readily available materials. Such

phosphatidyl, choline, cholesterol, phosphatidyl ethanolamine, materials include glycerol (DOPC), dioleoylphosphatidyl (DOPG), dioleoylphosphatidyl choline dioleoylphoshatidyl ethanolamine (DOPE), among others. These materials can also be mixed with the DOTMA and DOTAP starting materials in appropriate ratios. Methods for making liposomes using these materials are well known in the art.

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For example, commercially dioleoylphosphatidyl choline (DOPC), dioleoylphosphatidyl glycerol (DOPG), and dioleoylphosphatidyl ethanolamine (DOPE) can be used in various combinations to make conventional liposomes, with or without the addition of cholesterol. Thus, for example, DOPG/DOPC vesicles can be prepared by drying 50 mg each of DOPG and DOPC under a stream of nitrogen gas into a sonication vial. The sample is placed under a vacuum pump overnight and is hydrated the following day with deionized water. The sample is then sonicated for 2 hours in a capped vial, using a Heat Systems model 350 sonicator equipped with an inverted cup (bath type) probe at the maximum setting while the bath is circulated at 15EC. Alternatively, negatively charged vesicles can be prepared without sonication to produce multilamellar vesicles or by extrusion through nucleopore membranes to produce unilamellar vesicles of discrete size. Other methods are known and available to those of skill in the art.

The liposomes can comprise multilamellar vesicles (MLVs), small unilamellar vesicles (SUVs), or large unilamellar vesicles (LUVs), with SUVs being preferred. The various liposome-nucleic acid complexes are prepared using methods well known in the art. See, e.g., Straubinger et al., Methods of Immunology (1983), 101:512-527, which is herein incorporated by reference. For example, MLVs containing nucleic acid can be prepared by depositing a thin film of phospholipid on the walls of a glass tube and subsequently hydrating with a solution of the material to be encapsulated. SUVs are prepared by extended sonication of MLVs to produce a homogeneous population of unilamellar liposomes. The material to be entrapped is added to a suspension of preformed MLVs and then sonicated. When using liposomes containing cationic lipids, the dried lipid film is resuspended in an appropriate solution such as sterile water or an isotonic buffer solution such as 10 mM Tris/NaCl, sonicated, and then the preformed liposomes are mixed directly with the DNA. The liposome and DNA form a very stable complex due to binding of the positively charged liposomes to the cationic DNA. SUVs find use with small nucleic acid fragments. LUVs are prepared by a number of methods, well known in the art. Commonly used methods include Ca2+-EDTA chelation (Papahadjopoulos et al., Biochim. Biophys. Acta (1975) 394:483; Wilson et al., Cell 17:77 (1979)); ether injection (Deamer, D. and Bangham, A., Biochim. Biophys. Acta 443:629 (1976); Ostro et al., Biochem. Biophys. Res. Commun. 76:836 (1977); Fraley et al., Proc. Natl. Acad. Sci. USA 76:3348 (1979)); detergent dialysis (Enoch, H. and Strittmatter, P., Proc. Natl. Acad. Sci. USA 76:145 (1979)); and reverse-phase evaporation (REV) (Fraley et al., J. Biol. Chem. 255:10431 (1980); Szoka, F. and

Papahadjopoulos, D., Proc. Natl. Acad. Sci. USA 75:145 (1978); Schaefer-Ridder et al., Science 215:166 (1982)), which are herein incorporated by reference.

Generally, the ratio of DNA to liposomes will be from about 10:1 to about 1:10. Preferably, the ration will be from about 5:1 to about 1:5. More preferably, the ration will be about 3:1 to about 1:3. Still more preferably, the ratio will be about 1:1.

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U.S. Patent No. 5,676,954 (which is herein incorporated by reference) reports on the injection of genetic material, complexed with cationic liposomes carriers, into mice. U.S. Patent Nos. 4,897,355, 4,946,787, 5,049,386, 5,459,127, 5,589,466, 5,693,622, 5,580,859, 5,703,055, and international publication no. WO 94/9469 (which are herein incorporated by reference) provide cationic lipids for use in transfecting DNA into cells and mammals. U.S. Patent Nos. 5,589,466, 5,693,622, 5,580,859, 5,703,055, and international publication no. WO 94/9469 provide methods for delivering DNA-cationic lipid complexes to mammals.

In certain embodiments, cells are engineered, ex vivo or *in vivo*, using a retroviral particle containing RNA which comprises a sequence encoding a polypeptide of the present invention. Retroviruses from which the retroviral plasmid vectors may be derived include, but are not limited to, Moloney Murine Leukemia Virus, spleen necrosis virus, Rous sarcoma Virus, Harvey Sarcoma Virus, avian leukosis virus, gibbon ape leukemia virus, human immunodeficiency virus, Myeloproliferative Sarcoma Virus, and mammary tumor virus.

The retroviral plasmid vector is employed to transduce packaging cell lines to form producer cell lines. Examples of packaging cells which may be transfected include, but are not limited to, the PE501, PA317, R-2, R-AM, PA12, T19-14X, VT-19-17-H2, RCRE, RCRIP, GP+E-86, GP+envAm12, and DAN cell lines as described in Miller, Human Gene Therapy 1:5-14 (1990), which is incorporated herein by reference in its entirety. The vector may transduce the packaging cells through any means known in the art. Such means include, but are not limited to, electroporation, the use of liposomes, and CaPO₄ precipitation. In one alternative, the retroviral plasmid vector may be encapsulated into a liposome, or coupled to a lipid, and then administered to a host.

The producer cell line generates infectious retroviral vector particles which include polynucleotide encoding a polypeptide of the present invention. Such retroviral vector particles then may be employed, to transduce eukaryotic cells, either in vitro or *in vivo*. The transduced eukaryotic cells will express a polypeptide of the present invention.

In certain other embodiments, cells are engineered, ex vivo or *in vivo*, with polynucleotide contained in an adenovirus vector. Adenovirus can be manipulated such that it encodes and expresses a polypeptide of the present invention, and at the same time is inactivated in terms of its ability to replicate in a normal lytic viral life cycle. Adenovirus expression is achieved without integration of the viral DNA into the host cell chromosome, thereby alleviating concerns about insertional mutagenesis. Furthermore, adenoviruses have been used as live enteric vaccines for

many years with an excellent safety profile (Schwartz et al. Am. Rev. Respir. Dis.109:233-238 (1974)). Finally, adenovirus mediated gene transfer has been demonstrated in a number of instances including transfer of alpha-1-antitrypsin and CFTR to the lungs of cotton rats (Rosenfeld, M. A. et al. (1991) Science 252:431-434; Rosenfeld et al., (1992) Cell 68:143-155). Furthermore, extensive studies to attempt to establish adenovirus as a causative agent in human cancer were uniformly negative (Green, M. et al. (1979) Proc. Natl. Acad. Sci. USA 76:6606).

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Suitable adenoviral vectors useful in the present invention are described, for example, in Kozarsky and Wilson, Curr. Opin. Genet. Devel. 3:499-503 (1993); Rosenfeld et al., Cell 68:143-155 (1992); Engelhardt et al., Human Genet. Ther. 4:759-769 (1993); Yang et al., Nature Genet. 7:362-369 (1994); Wilson et al., Nature 365:691-692 (1993); and U.S. Patent No. 5,652,224, which are herein incorporated by reference. For example, the adenovirus vector Ad2 is useful and can be grown in human 293 cells. These cells contain the E1 region of adenovirus and constitutively express Ela and Elb, which complement the defective adenoviruses by providing the products of the genes deleted from the vector. In addition to Ad2, other varieties of adenovirus (e.g., Ad3, Ad5, and Ad7) are also useful in the present invention.

Preferably, the adenoviruses used in the present invention are replication deficient. Replication deficient adenoviruses require the aid of a helper virus and/or packaging cell line to form infectious particles. The resulting virus is capable of infecting cells and can express a polynucleotide of interest which is operably linked to a promoter, but cannot replicate in most cells. Replication deficient adenoviruses may be deleted in one or more of all or a portion of the following genes: E1a, E1b, E3, E4, E2a, or L1 through L5.

In certain other embodiments, the cells are engineered, ex vivo or *in vivo*, using an adeno-associated virus (AAV). AAVs are naturally occurring defective viruses that require helper viruses to produce infectious particles (Muzyczka, N., Curr. Topics in Microbiol. Immunol. 158:97 (1992)). It is also one of the few viruses that may integrate its DNA into non-dividing cells. Vectors containing as little as 300 base pairs of AAV can be packaged and can integrate, but space for exogenous DNA is limited to about 4.5 kb. Methods for producing and using such AAVs are known in the art. See, for example, U.S. Patent Nos. 5,139,941, 5,173,414, 5,354,678, 5,436,146, 5,474,935, 5,478,745, and 5,589,377.

For example, an appropriate AAV vector for use in the present invention will include all the sequences necessary for DNA replication, encapsidation, and host-cell integration. The polynucleotide construct is inserted into the AAV vector using standard cloning methods, such as those found in Sambrook et al., Molecular Cloning: A Laboratory Manual, Cold Spring Harbor Press (1989). The recombinant AAV vector is then transfected into packaging cells which are infected with a helper virus, using any standard technique, including lipofection, electroporation, calcium phosphate precipitation, etc. Appropriate helper viruses include adenoviruses, cytomegaloviruses, vaccinia viruses, or herpes viruses. Once the packaging cells are transfected

and infected, they will produce infectious AAV viral particles which contain the polynucleotide construct. These viral particles are then used to transduce eukaryotic cells, either ex vivo or in vivo. The transduced cells will contain the polynucleotide construct integrated into its genome, and will express a polypeptide of the invention.

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Another method of gene therapy involves operably associating heterologous control regions and endogenous polynucleotide sequences (e.g. encoding a polypeptide of the present invention) via homologous recombination (see, e.g., U.S. Patent No. 5,641,670, issued June 24, 1997; International Publication No. WO 96/29411, published September 26, 1996; International Publication No. WO 94/12650, published August 4, 1994; Koller et al., Proc. Natl. Acad. Sci. USA 86:8932-8935 (1989); and Zijlstra et al., Nature 342:435-438 (1989), which are herein encorporated by reference. This method involves the activation of a gene which is present in the target cells, but which is not normally expressed in the cells, or is expressed at a lower level than desired.

Polynucleotide constructs are made, using standard techniques known in the art, which contain the promoter with targeting sequences flanking the promoter. Suitable promoters are described herein. The targeting sequence is sufficiently complementary to an endogenous sequence to permit homologous recombination of the promoter-targeting sequence with the endogenous sequence. The targeting sequence will be sufficiently near the 5' end of the desired endogenous polynucleotide sequence so the promoter will be operably linked to the endogenous sequence upon homologous recombination.

The promoter and the targeting sequences can be amplified using PCR. Preferably, the amplified promoter contains distinct restriction enzyme sites on the 5' and 3' ends. Preferably, the 3' end of the first targeting sequence contains the same restriction enzyme site as the 5' end of the amplified promoter and the 5' end of the second targeting sequence contains the same restriction site as the 3' end of the amplified promoter. The amplified promoter and targeting sequences are digested and ligated together.

The promoter-targeting sequence construct is delivered to the cells, either as naked polynucleotide, or in conjunction with transfection-facilitating agents, such as liposomes, viral sequences, viral particles, whole viruses, lipofection, precipitating agents, etc., described in more detail above. The P promoter-targeting sequence can be delivered by any method, included direct needle injection, intravenous injection, topical administration, catheter infusion, particle accelerators, etc. The methods are described in more detail below.

The promoter-targeting sequence construct is taken up by cells. Homologous recombination between the construct and the endogenous sequence takes place, such that an endogenous sequence is placed under the control of the promoter. The promoter then drives the expression of the endogenous sequence.

The polynucleotide encoding a polypeptide of the present invention may contain a secretory signal sequence that facilitates secretion of the protein. Typically, the signal sequence is positioned in the coding region of the polynucleotide to be expressed towards or at the 5' end of the coding region. The signal sequence may be homologous or heterologous to the polynucleotide of interest and may be homologous or heterologous to the cells to be transfected. Additionally, the signal sequence may be chemically synthesized using methods known in the art.

Any mode of administration of any of the above-described polynucleotides constructs can be used so long as the mode results in the expression of one or more molecules in an amount sufficient to provide a therapeutic effect. This includes direct needle injection, systemic injection, catheter infusion, biolistic injectors, particle accelerators (i.e., "gene guns"), gelfoam sponge depots, other commercially available depot materials, osmotic pumps (e.g., Alza minipumps), oral or suppositorial solid (tablet or pill) pharmaceutical formulations, and decanting or topical applications during surgery. For example, direct injection of naked calcium phosphate-precipitated plasmid into rat liver and rat spleen or a protein-coated plasmid into the portal vein has resulted in gene expression of the foreign gene in the rat livers (Kaneda et al., Science 243:375 (1989)).

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A preferred method of local administration is by direct injection. Preferably, a recombinant molecule of the present invention complexed with a delivery vehicle is administered by direct injection into or locally within the area of arteries. Administration of a composition locally within the area of arteries refers to injecting the composition centimeters and preferably, millimeters within arteries.

Another method of local administration is to contact a polynucleotide construct of the present invention in or around a surgical wound. For example, a patient can undergo surgery and the polynucleotide construct can be coated on the surface of tissue inside the wound or the construct can be injected into areas of tissue inside the wound.

Therapeutic compositions useful in systemic administration, include recombinant molecules of the present invention complexed to a targeted delivery vehicle of the present invention. Suitable delivery vehicles for use with systemic administration comprise liposomes comprising ligands for targeting the vehicle to a particular site. In specific embodiments, suitable delivery vehicles for use with systemic administration comprise liposomes comprising polypeptides of the invention for targeting the vehicle to a particular site.

Preferred methods of systemic administration, include intravenous injection, aerosol, oral and percutaneous (topical) delivery. Intravenous injections can be performed using methods standard in the art. Aerosol delivery can also be performed using methods standard in the art (see, for example, Stribling et al., Proc. Natl. Acad. Sci. USA 189:11277-11281, 1992, which is incorporated herein by reference). Oral delivery can be performed by complexing a polynucleotide construct of the present invention to a carrier capable of withstanding degradation by digestive

enzymes in the gut of an animal. Examples of such carriers, include plastic capsules or tablets, such as those known in the art. Topical delivery can be performed by mixing a polynucleotide construct of the present invention with a lipophilic reagent (e.g., DMSO) that is capable of passing into the skin.

Determining an effective amount of substance to be delivered can depend upon a number of factors including, for example, the chemical structure and biological activity of the substance, the age and weight of the animal, the precise condition requiring treatment and its severity, and the route of administration. The frequency of treatments depends upon a number of factors, such as the amount of polynucleotide constructs administered per dose, as well as the health and history of the subject. The precise amount, number of doses, and timing of doses will be determined by the attending physician or veterinarian.

Therapeutic compositions of the present invention can be administered to any animal, preferably to mammals and birds. Preferred mammals include humans, dogs, cats, mice, rats, rabbits sheep, cattle, horses and pigs, with humans being particularly preferred.

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## **Biological Activities**

Polynucleotides or polypeptides, or agonists or antagonists of the present invention, can be used in assays to test for one or more biological activities. If these polynucleotides or polypeptides, or agonists or antagonists of the present invention, do exhibit activity in a particular assay, it is likely that these molecules may be involved in the diseases associated with the biological activity. Thus, the polynucleotides and polypeptides, and agonists or antagonists could be used to treat the associated disease.

Members of the secreted family of proteins are believed to be involved in biological activities associated with, for example, cellular signaling. Accordingly, compositions of the invention (including polynucleotides, polypeptides and antibodies of the invention, and fragments and variants thereof) may be used in diagnosis, prognosis, prevention and/or treatment of diseases and/or disorders associated with aberrant activity of secreted polypeptides.

In preferred embodiments, compositions of the invention (including polynucleotides, polypeptides and antibodies of the invention, and fragments and variants thereof) may be used in the diagnosis, prognosis, prevention, treatment, and/or amelioration of diseases and/or disorders relating to the hematopoietic system (e.g., blood coagulation disorders (e.g., hemophilia); blood clotting disorders (e.g., thromboembolism, and pulmonary embolism); fibrinolysis disorders; complement activation disorders (e.g., complement component deficiencies); hematopoietic disorders (e.g., X-linked agammaglobulinemia, anemia); or immune system disorders (e.g., autoimmunity and immunodeficiencies)); and/or as described under "Immune Activity", "Cardiovascular Disorders", and "Blood-Related Disorders" below, and neoplastic disorders (e.g.,

cell migration, prohormone activation, extracellular matrix turnover), and/or as described under "Immune Activity" and "Hyperproliferative Disorders" below.

In certain embodiments, a polypeptide of the invention, or polynucleotides, antibodies, agonists, or antagonists corresponding to that polypeptide, may be used to diagnose and/or prognosticate diseases and/or disorders associated with the tissue(s) in which the polypeptide of the invention is expressed including one, two, three, four, five, or more tissues disclosed in Table 1B.2, column 5 (Tissue Distribution Library Code).

Thus, polynucleotides, translation products and antibodies of the invention are useful in the diagnosis, detection, prevention, prognistication, and/or treatment of diseases and/or disorders associated with activities that include, but are not limited to, prohormone activation, neurotransmitter activity, cellular signaling, cellular proliferation, cellular differentiation, and cell migration.

More generally, polynucleotides, translation products and antibodies corresponding to this gene may be useful for the diagnosis, prognosis, prevention, treatment and/or amelioration of diseases and/or disorders associated with the following system or systems.

#### **Immune Activity**

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Polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in preventing, diagnosing, prognosticating, treating, and/or ameliorating diseases, disorders, and/or conditions of the immune system, by, for example, activating or inhibiting the proliferation, differentiation, or mobilization (chemotaxis) of immune cells. Immune cells develop through a process called hematopoiesis, producing myeloid (platelets, red blood cells, neutrophils, and macrophages) and lymphoid (B and T lymphocytes) cells from pluripotent stem cells. The etiology of these immune diseases, disorders, and/or conditions may be genetic, somatic, such as cancer and some autoimmune diseases, acquired (e.g., by chemotherapy or toxins), or infectious. Moreover, polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention can be used as a marker or detector of a particular immune system disease or disorder.

In another embodiment, a polypeptide of the invention, or polynucleotides, antibodies, agonists, or antagonists corresponding to that polypeptide, may be used to treat diseases and disorders of the immune system and/or to inhibit or enhance an immune response generated by cells associated with the tissue(s) in which the polypeptide of the invention is expressed, including

one, two, three, four, five, or more tissues disclosed in Table 1B.2, column 5 (Tissue Distribution Library Code).

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Polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in preventing, diagnosing, prognosticating, treating and/or ameliorating immunodeficiencies, including both congenital and acquired immunodeficiencies. Examples of B cell immunodeficiencies in which immunoglobulin levels B cell function and/or B cell numbers are decreased include: X-linked agammaglobulinemia (Bruton's disease), X-linked infantile agammaglobulinemia, X-linked immunodeficiency with hyper IgM, non X-linked immunodeficiency with hyper IgM, X-linked lymphoproliferative syndrome (XLP), agammaglobulinemia including congenital and acquired agammaglobulinemia, adult onset agammaglobulinemia, dysgammaglobulinemia, agammaglobulinemia, late-onset hypogammaglobulinemia, unspecified hypogammaglobulinemia, recessive agammaglobulinemia (Swiss type), Selective IgM deficiency, selective IgA deficiency, selective IgG subclass deficiencies, IgG subclass deficiency (with or without IgA deficiency), Ig deficiency with increased IgM, IgG and IgA deficiency with increased IgM, antibody deficiency with normal or elevated Igs, Ig heavy chain deletions, kappa chain deficiency, B cell lymphoproliferative disorder (BLPD), common variable immunodeficiency (CVID), common variable immunodeficiency (CVI) (acquired), and transient hypogammaglobulinemia of infancy.

In specific embodiments, ataxia-telangiectasia or conditions associated with ataxia-telangiectasia are detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated using the polypeptides or polynucleotides of the invention, and/or agonists or antagonists thereof.

Examples of congenital immunodeficiencies in which T cell and/or B cell function and/or number is decreased include, but are not limited to: DiGeorge anomaly, severe combined immunodeficiencies (SCID) (including, but not limited to, X-linked SCID, autosomal recessive SCID, adenosine dearninase deficiency, purine nucleoside phosphorylase (PNP) deficiency, Class II MHC deficiency (Bare lymphocyte syndrome), Wiskott-Aldrich syndrome, and ataxia telangiectasia), thymic hypoplasia, third and fourth pharyngeal pouch syndrome, 22q11.2 deletion, chronic mucocutaneous candidiasis, natural killer cell deficiency (NK), idiopathic CD4+ T-lymphocytopenia, immunodeficiency with predominant T cell defect (unspecified), and unspecified immunodeficiency of cell mediated immunity.

In specific embodiments, DiGeorge anomaly or conditions associated with DiGeorge anomaly are prevented, detected, diagnosed, prognosticated, treated and/or ameliorated using polypeptides or polynucleotides of the invention, or antagonists or agonists thereof.

Other immunodeficiencies that may be prevented, detected, diagnosed, prognosticated, treated and/or ameliorated using polypeptides or polynucleotides of the invention, and/or agonists or antagonists thereof, include, but are not limited to, chronic granulomatous disease, Chédiak-Higashi syndrome, myeloperoxidase deficiency, leukocyte glucose-6-phosphate dehydrogenase deficiency, X-linked lymphoproliferative syndrome (XLP), leukocyte adhesion deficiency, complement component deficiencies (including C1, C2, C3, C4, C5, C6, C7, C8 and/or C9 deficiencies), reticular dysgenesis, thymic alymphoplasia-aplasia, immunodeficiency with thymoma, severe congenital leukopenia, dysplasia with immunodeficiency, neonatal neutropenia, short limbed dwarfism, and Nezelof syndrome-combined immunodeficiency with Igs.

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In a preferred embodiment, the immunodeficiencies and/or conditions associated with the immunodeficiencies recited above are prevented, detected, diagnosed, prognosticated, treated and/or ameliorated using polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention.

In a preferred embodiment polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention could be used as an agent to boost immunoresponsiveness among immunodeficient individuals. In specific embodiments, polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention could be used as an agent to boost immunoresponsiveness among B cell and/or T cell immunodeficient individuals.

The polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in preventing, detecting, diagnosing, prognosticating, treating and/or ameliorating autoimmune disorders. Many autoimmune disorders result from inappropriate recognition of self as foreign material by immune cells. This inappropriate recognition results in an immune response leading to the destruction of the host tissue. Therefore, the administration of polynucleotides and polypeptides of the invention that can inhibit an immune response, particularly the proliferation, differentiation, or chemotaxis of T-cells, may be an effective therapy in preventing autoimmune disorders.

Autoimmune diseases or disorders that may be prevented, detected, diagnosed, prognosticated, treated, and/or ameliorated by polynucleotides, polypeptides, antibodies, and/or

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agonists or antagonists of the present invention include, but are not limited to, one or more of the following: systemic lupus erythematosus, rheumatoid arthritis, ankylosing spondylitis, multiple sclerosis, autoimmune thyroiditis, Hashimoto's thyroiditis, autoimmune hemolytic anemia, hemolytic anemia, thrombocytopenia, autoimmune thrombocytopenia purpura, autoimmune neonatal thrombocytopenia, idiopathic thrombocytopenia purpura, purpura (e.g., Henloch-Scoenlein purpura), autoimmunocytopenia, Goodpasture's syndrome, Pemphigus vulgaris, myasthenia gravis, Grave's disease (hyperthyroidism), and insulin-resistant diabetes mellitus.

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Additional disorders that are likely to have an autoimmune component that may be prevented, detected, diagnosed, prognosticated, treated and/or ameliorated with the compositions of the invention include, but are not limited to, type II collagen-induced arthritis, antiphospholipid syndrome, dermatitis, allergic encephalomyelitis, myocarditis, relapsing polychondritis, rheumatic heart disease, neuritis, uveitis ophthalmia, polyendocrinopathies, Reiter's Disease, Stiff-Man Syndrome, autoimmune pulmonary inflammation, autism, Guillain-Barre Syndrome, insulin dependent diabetes mellitus, and autoimmune inflammatory eye disorders.

Additional disorders that are likely to have an autoimmune component that may be prevented, detected, diagnosed, prognosticated, treated and/or ameliorated with the compositions of the invention include, but are not limited to, scleroderma with anti-collagen antibodies (often characterized, e.g., by nucleolar and other nuclear antibodies), mixed connective tissue disease (often characterized, e.g., by antibodies to extractable nuclear antigens (e.g., ribonucleoprotein)), polymyositis (often characterized, e.g., by nonhistone ANA), pernicious anemia (often characterized, e.g., by antiparietal cell, microsomes, and intrinsic factor antibodies), idiopathic Addison's disease (often characterized, e.g., by humoral and cell-mediated adrenal cytotoxicity, infertility (often characterized, e.g., by antispermatozoal antibodies), glomerulonephritis (often characterized, e.g., by glomerular basement membrane antibodies or immune complexes), bullous pemphigoid (often characterized, e.g., by IgG and complement in basement membrane), Sjogren's syndrome (often characterized, e.g., by multiple tissue antibodies, and/or a specific nonhistone ANA (SS-B)), diabetes mellitus (often characterized, e.g., by cell-mediated and humoral islet cell antibodies), and adrenergic drug resistance (including adrenergic drug resistance with asthma or cystic fibrosis) (often characterized, e.g., by beta-adrenergic receptor antibodies).

Additional disorders that may have an autoimmune component that may be prevented, detected, diagnosed, prognosticated, treated and/or ameliorated with the compositions of the

invention include, but are not limited to, chronic active hepatitis (often characterized, e.g., by smooth muscle antibodies), primary biliary cirrhosis (often characterized, e.g., by mitochondria antibodies), other endocrine gland failure (often characterized, e.g., by specific tissue antibodies in some cases), vitiligo (often characterized, e.g., by melanocyte antibodies), vasculitis (often characterized, e.g., by Ig and complement in vessel walls and/or low serum complement), post-MI (often characterized, e.g., by myocardial antibodies), cardiotomy syndrome (often characterized, e.g., by myocardial antibodies), urticaria (often characterized, e.g., by IgG and IgM antibodies to IgE), atopic dermatitis (often characterized, e.g., by IgG and IgM antibodies to IgE), asthma (often characterized, e.g., by IgG and IgM antibodies to IgE), and many other inflammatory, granulomatous, degenerative, and atrophic disorders.

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In a preferred embodiment, the autoimmune diseases and disorders and/or conditions associated with the diseases and disorders recited above are prevented, detected, diagnosed, prognosticated, treated and/or ameliorated using for example, antagonists or agonists, polypeptides or polynucleotides, or antibodies of the present invention. In a specific preferred embodiment, rheumatoid arthritis is prevented, detected, diagnosed, prognosticated, treated and/or ameliorated using polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention.

In another specific preferred embodiment, systemic lupus erythematosus is prevented, detected, diagnosed, prognosticated, treated and/or ameliorated using polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention. In another specific preferred embodiment, idiopathic thrombocytopenia purpura is prevented, detected, diagnosed, prognosticated, treated and/or ameliorated using polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention.

In another specific preferred embodiment IgA nephropathy is prevented, detected, diagnosed, prognosticated, treated and/or ameliorated using polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention.

In a preferred embodiment, the autoimmune diseases and disorders and/or conditions associated with the diseases and disorders recited above are prevented, detected, diagnosed, prognosticated, treated and/or ameliorated using polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention

In preferred embodiments, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a immunosuppressive agent(s).

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Polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating diseases, disorders, and/or conditions of hematopoietic cells. Polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention could be used to increase differentiation and proliferation of hematopoietic cells, including the pluripotent stem cells, in an effort to treat or prevent those diseases, disorders, and/or conditions associated with a decrease in certain (or many) types hematopoietic cells, including but not limited to, leukopenia, neutropenia, anemia, and thrombocytopenia. Alternatively, Polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention could be used to increase differentiation and proliferation of hematopoietic cells, including the pluripotent stem cells, in an effort to treat or prevent those diseases, disorders, and/or conditions associated with an increase in certain (or many) types of hematopoietic cells, including but not limited to, histiocytosis.

Allergic reactions and conditions, such as asthma (particularly allergic asthma) or other respiratory problems, may also be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated using polypeptides, antibodies, or polynucleotides of the invention, and/or agonists or antagonists thereof. Moreover, these molecules can be used to treat, prevent, prognose, and/or diagnose anaphylaxis, hypersensitivity to an antigenic molecule, or blood group incompatibility.

Additionally, polypeptides or polynucleotides of the invention, and/or agonists or antagonists thereof, may be used to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate IgE-mediated allergic reactions. Such allergic reactions include, but are not limited to, asthma, rhinitis, and eczema. In specific embodiments, polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be used to modulate IgE concentrations in vitro or in vivo.

Moreover, polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention have uses in the detection, prevention, diagnosis, prognostication, treatment, and/or amelioration of inflammatory conditions. For example, since polypeptides, antibodies, or polynucleotides of the invention, and/or agonists or antagonists of the invention may inhibit the activation, proliferation and/or differentiation of cells involved in an inflammatory response, these molecules can be used to prevent and/or treat chronic and acute inflammatory conditions. Such

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inflammatory conditions include, but are not limited to, for example, inflammation associated with infection (e.g., septic shock, sepsis, or systemic inflammatory response syndrome), ischemia-reperfusion injury, endotoxin lethality, complement-mediated hyperacute rejection, nephritis, cytokine or chemokine induced lung injury, inflammatory bowel disease, Crohn's disease, over production of cytokines (e.g., TNF or IL-1.), respiratory disorders (e.g., asthma and allergy); gastrointestinal disorders (e.g., inflammatory bowel disease); cancers (e.g., gastric, ovarian, lung, bladder, liver, and breast); CNS disorders (e.g., multiple sclerosis; ischemic brain injury and/or stroke, traumatic brain injury, neurodegenerative disorders (e.g., Parkinson's disease and Alzheimer's disease); AIDS-related dementia; and prion disease); cardiovascular disorders (e.g., atherosclerosis, myocarditis, cardiovascular disease, and cardiopulmonary bypass complications); as well as many additional diseases, conditions, and disorders that are characterized by inflammation (e.g., hepatitis, rheumatoid arthritis, gout, trauma, pancreatitis, sarcoidosis, dermatitis, renal ischemia-reperfusion injury, Grave's disease, systemic lupus erythematosus, diabetes mellitus, and allogenic transplant rejection).

Because inflammation is a fundamental defense mechanism, inflammatory disorders can effect virtually any tissue of the body. Accordingly, polynucleotides, polypeptides, and antibodies of the invention, as well as agonists or antagonists thereof, have uses in the treatment of tissue-specific inflammatory disorders, including, but not limited to, adrenalitis, alveolitis, angiocholecystitis, appendicitis, balanitis, blepharitis, bronchitis, bursitis, carditis, cellulitis, cervicitis, cholecystitis, chorditis, cochlitis, colitis, conjunctivitis, cystitis, dermatitis, diverticulitis, encephalitis, endocarditis, esophagitis, eustachitis, fibrositis, folliculitis, gastritis, gastroenteritis, gingivitis, glossitis, hepatosplenitis, keratitis, labyrinthitis, laryngitis, lymphangitis, mastitis, media otitis, meningitis, metritis, mucitis, myocarditis, myosititis, myringitis, nephritis, neuritis, orchitis, osteochondritis, otitis, pericarditis, peritendonitis, peritonitis, pharyngitis, phlebitis, poliomyelitis, prostatitis, pulpitis, retinitis, rhinitis, salpingitis, scleritis, sclerochoroiditis, urethritis, and vaginitis.

In specific embodiments, polypeptides, antibodies, or polynucleotides of the invention, and/or agonists or antagonists thereof, are useful to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate organ transplant rejections and graft-versus-host disease. Organ rejection occurs by host immune cell destruction of the transplanted tissue through an immune response. Similarly,

an immune response is also involved in GVHD, but, in this case, the foreign transplanted immune cells destroy the host tissues. Polypeptides, antibodies, or polynucleotides of the invention, and/or agonists or antagonists thereof, that inhibit an immune response, particularly the activation, proliferation, differentiation, or chemotaxis of T-cells, may be an effective therapy in preventing organ rejection or GVHD. In specific embodiments, polypeptides, antibodies, or polynucleotides of the invention, and/or agonists or antagonists thereof, that inhibit an immune response, particularly the activation, proliferation, differentiation, or chemotaxis of T-cells, may be an effective therapy in preventing experimental allergic and hyperacute xenograft rejection.

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In other embodiments, polypeptides, antibodies, or polynucleotides of the invention, and/or agonists or antagonists thereof, are useful to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate immune complex diseases, including, but not limited to, serum sickness, post streptococcal glomerulonephritis, polyarteritis nodosa, and immune complex-induced vasculitis.

Polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the invention can be used to treat, detect, and/or prevent infectious agents. For example, by increasing the immune response, particularly increasing the proliferation activation and/or differentiation of B and/or T cells, infectious diseases may be treated, detected, and/or prevented. The immune response may be increased by either enhancing an existing immune response, or by initiating a new immune response. Alternatively, polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may also directly inhibit the infectious agent (refer to section of application listing infectious agents, etc), without necessarily eliciting an immune response.

In another embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a vaccine adjuvant that enhances immune responsiveness to an antigen. In a specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an adjuvant to enhance tumor-specific immune responses.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an adjuvant to enhance anti-viral immune responses. Anti-viral immune responses that may be enhanced using the compositions of the invention as an adjuvant, include virus and virus associated diseases or symptoms described herein or otherwise known in the art. In specific embodiments, the compositions of the invention are

used as an adjuvant to enhance an immune response to a virus, disease, or symptom selected from the group consisting of: AIDS, meningitis, Dengue, EBV, and hepatitis (e.g., hepatitis B). In another specific embodiment, the compositions of the invention are used as an adjuvant to enhance an immune response to a virus, disease, or symptom selected from the group consisting of: HIV/AIDS, respiratory syncytial virus, Dengue, rotavirus, Japanese B encephalitis, influenza A and B, parainfluenza, measles, cytomegalovirus, rabies, Junin, Chikungunya, Rift Valley Fever, herpes simplex, and yellow fever.

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In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an adjuvant to enhance anti-bacterial or antifungal immune responses. Anti-bacterial or anti-fungal immune responses that may be enhanced using the compositions of the invention as an adjuvant, include bacteria or fungus and bacteria or fungus associated diseases or symptoms described herein or otherwise known in the art. In specific embodiments, the compositions of the invention are used as an adjuvant to enhance an immune response to a bacteria or fungus, disease, or symptom selected from the group consisting of: tetanus, Diphtheria, botulism, and meningitis type B.

In another specific embodiment, the compositions of the invention are used as an adjuvant to enhance an immune response to a bacteria or fungus, disease, or symptom selected from the group consisting of: Vibrio cholerae, Mycobacterium leprae, Salmonella typhi, Salmonella paratyphi, Meisseria meningitidis, Streptococcus pneumoniae, Group B streptococcus, Shigella spp., Enterotoxigenic Escherichia coli, Enterohemorrhagic E. coli, and Borrelia burgdorferi.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an adjuvant to enhance anti-parasitic immune responses. Anti-parasitic immune responses that may be enhanced using the compositions of the invention as an adjuvant, include parasite and parasite associated diseases or symptoms described herein or otherwise known in the art. In specific embodiments, the compositions of the invention are used as an adjuvant to enhance an immune response to a parasite. In another specific embodiment, the compositions of the invention are used as an adjuvant to enhance an immune response to Plasmodium (malaria) or Leishmania.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention may also be employed to treat infectious diseases including

silicosis, sarcoidosis, and idiopathic pulmonary fibrosis; for example, by preventing the recruitment and activation of mononuclear phagocytes.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an antigen for the generation of antibodies to inhibit or enhance immune mediated responses against polypeptides of the invention.

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In one embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are administered to an animal (e.g., mouse, rat, rabbit, hamster, guinea pig, pigs, micro-pig, chicken, camel, goat, horse, cow, sheep, dog, cat, non-human primate, and human, most preferably human) to boost the immune system to produce increased quantities of one or more antibodies (e.g., IgG, IgA, IgM, and IgE), to induce higher affinity antibody production and immunoglobulin class switching (e.g., IgG, IgA, IgM, and IgE), and/or to increase an immune response.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a stimulator of B cell responsiveness to pathogens.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an activator of T cells.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an agent that elevates the immune status of an individual prior to their receipt of immunosuppressive therapies.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an agent to induce higher affinity antibodies.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an agent to increase serum immunoglobulin concentrations.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an agent to accelerate recovery of immunocompromised individuals.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an agent to boost immunoresponsiveness among aged populations and/or neonates.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an immune system enhancer prior to, during, or after bone marrow transplant and/or other transplants (e.g., allogeneic or xenogeneic organ transplantation). With respect to transplantation, compositions of the invention may be administered prior to, concomitant with, and/or after transplantation. In a specific embodiment, compositions of the invention are administered after transplantation, prior to the beginning of recovery of T-cell populations. In another specific embodiment, compositions of the invention are first administered after transplantation after the beginning of recovery of T cell populations, but prior to full recovery of B cell populations.

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In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an agent to boost immunoresponsiveness among individuals having an acquired loss of B cell function. Conditions resulting in an acquired loss of B cell function that may be ameliorated or treated by administering the polypeptides, antibodies, polynucleotides and/or agonists or antagonists thereof, include, but are not limited to, HIV Infection, AIDS, bone marrow transplant, and B cell chronic lymphocytic leukemia (CLL).

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an agent to boost immunoresponsiveness among individuals having a temporary immune deficiency. Conditions resulting in a temporary immune deficiency that may be ameliorated or treated by administering the polypeptides, antibodies, polynucleotides and/or agonists or antagonists thereof, include, but are not limited to, recovery from viral infections (e.g., influenza), conditions associated with malnutrition, recovery from infectious mononucleosis, or conditions associated with stress, recovery from measles, recovery from blood transfusion, and recovery from surgery.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a regulator of antigen presentation by monocytes, dendritic cells, and/or B-cells. In one embodiment, polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention enhance antigen presentation or antagonizes antigen presentation in vitro or in vivo. Moreover, in related embodiments, said enhancement or antagonism of antigen presentation may be useful as an anti-tumor treatment or to modulate the immune system.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an agent to direct an individual's immune system towards development of a humoral response (i.e. TH2) as opposed to a TH1 cellular response.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a means to induce tumor proliferation and thus make it more susceptible to anti-neoplastic agents. For example, multiple myeloma is a slowly dividing disease and is thus refractory to virtually all anti-neoplastic regimens. If these cells were forced to proliferate more rapidly their susceptibility profile would likely change.

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In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a stimulator of B cell production in pathologies such as AIDS, chronic lymphocyte disorder and/or Common Variable Immunodificiency.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a therapy for generation and/or regeneration of lymphoid tissues following surgery, trauma or genetic defect. In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used in the pretreatment of bone marrow samples prior to transplant.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a gene-based therapy for genetically inherited disorders resulting in immuno-incompetence/immunodeficiency such as observed among SCID patients.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a means of activating monocytes/macrophages to defend against parasitic diseases that effect monocytes such as Leishmania.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a means of regulating secreted cytokines that are elicited by polypeptides of the invention.

In another embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used in one or more of the applications decribed herein, as they may apply to veterinary medicine.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a means of blocking various aspects of immune responses to foreign agents or self. Examples of diseases or conditions in which blocking of certain aspects of immune responses may be desired include autoimmune disorders such as lupus, and arthritis, as well as immunoresponsiveness to skin allergies, inflammation, bowel disease, injury and diseases/disorders associated with pathogens.

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In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a therapy for preventing the B cell proliferation and Ig secretion associated with autoimmune diseases such as idiopathic thrombocytopenic purpura, systemic lupus erythematosus and multiple sclerosis.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a inhibitor of B and/or T cell migration in endothelial cells. This activity disrupts tissue architecture or cognate responses and is useful, for example in disrupting immune responses, and blocking sepsis.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a therapy for chronic hypergammaglobulinemia evident in such diseases as monoclonal gammopathy of undetermined significance (MGUS), Waldenstrom's disease, related idiopathic monoclonal gammopathies, and plasmacytomas.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention may be employed for instance to inhibit polypeptide chemotaxis and activation of macrophages and their precursors, and of neutrophils, basophils, B lymphocytes and some T-cell subsets, e.g., activated and CD8 cytotoxic T cells and natural killer cells, in certain autoimmune and chronic inflammatory and infective diseases. Examples of autoimmune diseases are described herein and include multiple sclerosis, and insulin-dependent diabetes.

The polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention may also be employed to treat idiopathic hyper-eosinophilic syndrome by, for example, preventing eosinophil production and migration.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used to enhance or inhibit complement mediated cell lysis.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used to enhance or inhibit antibody dependent cellular cytotoxicity.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention may also be employed for treating atherosclerosis, for example, by preventing monocyte infiltration in the artery wall.

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In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention may be employed to treat adult respiratory distress syndrome (ARDS).

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention may be useful for stimulating wound and tissue repair, stimulating angiogenesis, and/or stimulating the repair of vascular or lymphatic diseases or disorders. Additionally, agonists and antagonists of the invention may be used to stimulate the regeneration of mucosal surfaces.

In a specific embodiment, polynucleotides or polypeptides, and/or agonists thereof are used to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate a disorder characterized by primary or acquired immunodeficiency, deficient serum immunoglobulin production, recurrent infections, and/or immune system dysfunction. Moreover, polynucleotides or polypeptides, and/or agonists thereof may be used to treat or prevent infections of the joints, bones, skin, and/or parotid glands, blood-borne infections (e.g., sepsis, meningitis, septic arthritis, and/or osteomyelitis), autoimmune diseases (e.g., those disclosed herein), inflammatory disorders, and malignancies, and/or any disease or disorder or condition associated with these infections, diseases, disorders and/or malignancies) including, but not limited to, CVID, other primary immune deficiencies, HIV disease, CLL, recurrent bronchitis, sinusitis, otitis media, conjunctivitis, pneumonia, hepatitis, meningitis, herpes zoster (e.g., severe herpes zoster), and/or pneumocystis carnii. Other diseases and disorders that may be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated with polynucleotides or polypeptides, and/or agonists of the present invention include, but are not limited to, HIV infection, HTLV-BLV infection, lymphopenia, phagocyte bactericidal dysfunction anemia, thrombocytopenia, and hemoglobinuria.

In another embodiment, polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention are used to treat, and/or diagnose an individual having

common variable immunodeficiency disease ("CVID"; also known as "acquired agammaglobulinemia" and "acquired hypogammaglobulinemia") or a subset of this disease.

In a specific embodiment, polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be used to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate cancers or neoplasms including immune cell or immune tissue-related cancers or neoplasms. Examples of cancers or neoplasms that may be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated by polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention include, but are not limited to, acute myelogenous leukemia, chronic myelogenous leukemia, Hodgkin's disease, non-Hodgkin's lymphoma, acute lymphocytic anemia (ALL) Chronic lymphocyte leukemia, plasmacytomas, multiple myeloma, Burkitt's lymphoma, EBV-transformed diseases, and/or diseases and disorders described in the section entitled "Hyperproliferative Disorders" elsewhere herein.

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In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a therapy for decreasing cellular proliferation of Large B-cell Lymphomas.

In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as a means of decreasing the involvement of B cells and Ig associated with Chronic Myelogenous Leukemia.

In specific embodiments, the compositions of the invention are used as an agent to boost immunoresponsiveness among B cell immunodeficient individuals, such as, for example, an individual who has undergone a partial or complete splenectomy.

Antagonists of the invention include, for example, binding and/or inhibitory antibodies, antisense nucleic acids, ribozymes or soluble forms of the polypeptides of the present invention (e.g., Fc fusion protein; see, e.g., Example 9). Agonists of the invention include, for example, binding or stimulatory antibodies, and soluble forms of the polypeptides (e.g., Fc fusion proteins; see, e.g., Example 9). polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention may be employed in a composition with a pharmaceutically acceptable carrier, e.g., as described herein.

In another embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are administered to an animal (including, but not limited to, those listed above, and also including transgenic animals) incapable of producing functional

endogenous antibody molecules or having an otherwise compromised endogenous immune system, but which is capable of producing human immunoglobulin molecules by means of a reconstituted or partially reconstituted immune system from another animal (see, e.g., published PCT Application Nos. WO98/24893, WO/9634096, WO/9633735, and WO/9110741). Administration of polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention to such animals is useful for the generation of monoclonal antibodies against the polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention.

## 10 Blood-Related Disorders

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The polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be used to modulate hemostatic (the stopping of bleeding) or thrombolytic (clot dissolving) activity. For example, by increasing hemostatic or thrombolytic activity, polynucleotides or polypeptides, and/or agonists or antagonists of the present invention could be used to treat or prevent blood coagulation diseases, disorders, and/or conditions (e.g., afibrinogenemia, factor deficiencies, hemophilia), blood platelet diseases, disorders, and/or conditions (e.g., thrombocytopenia), or wounds resulting from trauma, surgery, or other causes. Alternatively, polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention that can decrease hemostatic or thrombolytic activity could be used to inhibit or dissolve clotting. These molecules could be important in the treatment or prevention of heart attacks (infarction), strokes, or scarring.

In specific embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be used to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate thrombosis, arterial thrombosis, venous thrombosis, thromboembolism, pulmonary embolism, atherosclerosis, myocardial infarction, transient ischemic attack, unstable angina. In specific embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be used for the prevention of occulsion of saphenous grafts, for reducing the risk of periprocedural thrombosis as might accompany angioplasty procedures, for reducing the risk of stroke in patients with atrial fibrillation including nonrheumatic atrial fibrillation, for reducing the risk of embolism associated with mechanical heart valves and or mitral valves disease. Other uses for the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention, include, but are not limited to, the prevention of occlusions in extrcorporeal devices (e.g., intravascular canulas, vascular access shunts in hemodialysis patients, hemodialysis machines, and cardiopulmonary bypass machines).

In another embodiment, a polypeptide of the invention, or polynucleotides, antibodies, agonists, or antagonists corresponding to that polypeptide, may be used to detect, prevent,

diagnose, prognosticate, treat, and/or ameliorate diseases and disorders of the blood and/or blood forming organs associated with the tissue(s) in which the polypeptide of the invention is expressed, including one, two, three, four, five, or more tissues disclosed in Table 1B.2, column 5 (Tissue Distribution Library Code).

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The polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be used to modulate hematopoietic activity (the formation of blood cells). For example, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be used to increase the quantity of all or subsets of blood cells, such as, for example, erythrocytes, lymphocytes (B or T cells), myeloid cells (e.g., basophils, eosinophils, neutrophils, mast cells, macrophages) and platelets. The ability to decrease the quantity of blood cells or subsets of blood cells may be useful in the detection, prevention, diagnosis, prognostication, treatment, and/or amelioration of anemias and leukopenias described below. Alternatively, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be used to decrease the quantity of all or subsets of blood cells, such as, for example, erythrocytes, lymphocytes (B or T cells), myeloid cells (e.g., basophils, eosinophils, neutrophils, mast cells, macrophages) and platelets. The ability to decrease the quantity of blood cells or subsets of blood cells may be useful in the detection, prevention, diagnosis, prognostication, treatment, and/or amelioration of leukocytoses, such as, for example eosinophilia.

The polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be used to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate blood dyscrasia.

Anemias are conditions in which the number of red blood cells or amount of hemoglobin (the protein that carries oxygen) in them is below normal. Anemia may be caused by excessive bleeding, decreased red blood cell production, or increased red blood cell destruction (hemolysis). The polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating anemias. Anemias that may be treated detect, prevented, diagnosed, prognosticated, treated, and/or ameliorated by the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention include iron deficiency anemia, hypochromic anemia, microcytic anemia, chlorosis, hereditary siderob; astic anemia, idiopathic acquired sideroblastic anemia, red cell aplasia, megaloblastic anemia (e.g., pernicious anemia, (vitamin B12 deficiency) and folic acid deficiency anemia), aplastic anemia, hemolytic anemias (e.g., autoimmune helolytic anemia, microangiopathic hemolytic anemia, and paroxysmal noctumal hemoglobinuria). The polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating anemias associated with diseases including but not limited to, anemias associated with systemic lupus erythematosus, cancers, lymphomas, chronic renal disease, and enlarged spleens. The

polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating anemias arising from drug treatments such as anemias associated with methyldopa, dapsone, and/or sulfadrugs. Additionally, rhe polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating anemias associated with abnormal red blood cell architecture including but not limited to, hereditary spherocytosis, hereditary elliptocytosis, glucose-6-phosphate dehydrogenase deficiency, and sickle cell anemia.

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The polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating hemoglobin abnormalities, (e.g., those associated with sickle cell anemia, hemoglobin C disease, hemoglobin S-C disease, and hemoglobin E disease). Additionally, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating thalassemias, including, but not limited to major and minor forms of alpha-thalassemia and beta-thalassemia.

In another embodiment, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating bleeding disorders including, but not limited to, thrombocytopenia (e.g., idiopathic thrombocytopenic purpura, and thrombotic thrombocytopenic purpura), Von Willebrand's disease, hereditary platelet disorders (e.g., storage pool disease such as Chediak-Higashi and Hermansky-Pudlak syndromes, thromboxane A2 dysfunction, thromboasthenia, and Bernard-Soulier syndrome), hemolytic-uremic syndrome, hemophelias such as hemophelia A or Factor VII deficiency and Christmas disease or Factor IX deficiency, Hereditary Hemorhhagic Telangiectsia, also known as Rendu-Osler-Weber syndrome, allergic purpura (Henoch Schonlein purpura) and disseminated intravascular coagulation.

The effect of the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention on the clotting time of blood may be monitored using any of the clotting tests known in the art including, but not limited to, whole blood partial thromboplastin time (PTT), the activated partial thromboplastin time (aPTT), the activated clotting time (ACT), the recalcified activated clotting time, or the Lee-White Clotting time.

Several diseases and a variety of drugs can cause platelet dysfunction. Thus, in a specific embodiment, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating acquired platelet dysfunction such as platelet dysfunction accompanying kidney failure, leukemia, multiple myeloma, cirrhosis of the liver, and systemic lupus erythematosus as well as platelet dysfunction associated with drug treatments, including treatment

with aspirin, ticlopidine, nonsteroidal anti-inflammatory drugs (used for arthritis, pain, and sprains), and penicillin in high doses.

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In another embodiment, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating diseases and disorders characterized by or associated with increased or decreased numbers of white blood cells. Leukopenia occurs when the number of white blood cells decreases below normal. Leukopenias include, but are not limited to, neutropenia and lymphocytopenia. An increase in the number of white blood cells compared to normal is known as leukocytosis. The body generates increased numbers of white blood cells during infection. Thus, leukocytosis may simply be a normal physiological parameter that reflects infection. Alternatively, leukocytosis may be an indicator of injury or other disease such as cancer. Leokocytoses, include but are not limited to, eosinophilia, and accumulations of macrophages. In specific embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating leukopenia. In other specific embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating leukocytosis.

Leukopenia may be a generalized decreased in all types of white blood cells, or may be a specific depletion of particular types of white blood cells. Thus, in specific embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating decreases in neutrophil numbers, known as neutropenia. Neutropenias that may be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated by the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention include, but are not limited to, infantile genetic agranulocytosis, familial neutropenia, cyclic neutropenia, neutropenias resulting from or associated with dietary deficiencies (e.g., vitamin B 12 deficiency or folic acid deficiency), neutropenias resulting from or associated with drug treatments (e.g., antibiotic regimens such as penicillin treatment, sulfonamide treatment, anticoagulant treatment, anticonvulsant drugs, anti-thyroid drugs, and cancer chemotherapy), and neutropenias resulting from increased neutrophil destruction that may occur in association with some bacterial or viral infections, allergic disorders, autoimmune diseases, conditions in which an individual has an enlarged spleen (e.g., Felty syndrome, malaria and sarcoidosis), and some drug treatment regimens.

The polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating lymphocytopenias (decreased numbers of B and/or T lymphocytes), including,

but not limited lymphocytopenias resulting from or associated with stress, drug treatments (e.g., drug treatment with corticosteroids, cancer chemotherapies, and/or radiation therapies), AIDS infection and/or other diseases such as, for example, cancer, rheumatoid arthritis, systemic lupus erythematosus, chronic infections, some viral infections and/or hereditary disorders (e.g., DiGeorge syndrome, Wiskott-Aldrich Syndome, severe combined immunodeficiency, ataxia telangiectsia).

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The polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating diseases and disorders associated with macrophage numbers and/or macrophage function including, but not limited to, Gaucher's disease, Niemann-Pick disease, Letterer-Siwe disease and Hand-Schuller-Christian disease.

In another embodiment, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating diseases and disorders associated with eosinophil numbers and/or eosinophil function including, but not limited to, idiopathic hypereosinophilic syndrome, eosinophilia-myalgia syndrome, and Hand-Schuller-Christian disease.

In yet another embodiment, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating leukemias and lymphomas including, but not limited to, acute lymphocytic (lymphpblastic) leukemia (ALL), acute myeloid (myelocytic, myelogenous, myeloblastic, or myelomonocytic) leukemia, chronic lymphocytic leukemia (e.g., B cell leukemias, T cell leukemias, Sezary syndrome, and Hairy cell leukemia), chronic myelocytic (myeloid, myelogenous, or granulocytic) leukemia, Hodgkin's lymphoma, non-hodgkin's lymphoma, Burkitt's lymphoma, and mycosis fungoides.

In other embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating diseases and disorders of plasma cells including, but not limited to, plasma cell dyscrasias, monoclonal gammaopathies, monoclonal gammopathies of undetermined significance, multiple myeloma, macroglobulinemia, Waldenstrom's macroglobulinemia, cryoglobulinemia, and Raynaud's phenomenon.

In other embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating myeloproliferative disorders, including but not limited to, polycythemia vera, relative polycythemia, secondary polycythemia, myelofibrosis, acute myelofibrosis, agnogenic myelod metaplasia, thrombocythemia, (including both primary and seconday thrombocythemia) and chronic myelocytic leukemia.

In other embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful as a treatment prior to surgery, to increase blood cell production.

In other embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful as an agent to enhance the migration, phagocytosis, superoxide production, antibody dependent cellular cytotoxicity of neutrophils, eosionophils and macrophages.

In other embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful as an agent to increase the number of stem cells in circulation prior to stem cells pheresis. In another specific embodiment, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful as an agent to increase the number of stem cells in circulation prior to platelet pheresis.

In other embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful as an agent to increase cytokine production.

In other embodiments, the polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention may be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating primary hematopoietic and hematologic disorders.

## 20 Hyperproliferative Disorders

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In certain embodiments, polynucleotides or polypeptides, or agonists or antagonists of the present invention can be used to treat or detect hyperproliferative disorders, including neoplasms. Polynucleotides or polypeptides, or agonists or antagonists of the present invention may inhibit the proliferation of the disorder through direct or indirect interactions. Alternatively, Polynucleotides or polypeptides, or agonists or antagonists of the present invention may proliferate other cells which can inhibit the hyperproliferative disorder.

For example, by increasing an immune response, particularly increasing antigenic qualities of the hyperproliferative disorder or by proliferating, differentiating, or mobilizing T-cells, hyperproliferative disorders can be treated. This immune response may be increased by either enhancing an existing immune response, or by initiating a new immune response. Alternatively, decreasing an immune response may also be a method of treating hyperproliferative disorders, such as a chemotherapeutic agent.

Examples of hyperproliferative disorders that can be treated or detected by polynucleotides or polypeptides, or agonists or antagonists of the present invention include, but are not limited to neoplasms located in the: colon, abdomen, bone, breast, digestive system, liver, pancreas, peritoneum, endocrine glands (adrenal, parathyroid, pituitary, testicles, ovary, thymus,

thyroid), eye, head and neck, nervous (central and peripheral), lymphatic system, pelvis, skin, soft tissue, spleen, thorax, and urogenital tract.

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Similarly, other hyperproliferative disorders can also be treated or detected by polynucleotides or polypeptides, or agonists or antagonists of the present invention. Examples of such hyperproliferative disorders include, but are not limited to: Acute Childhood Lymphoblastic Leukemia, Acute Lymphoblastic Leukemia, Acute Lymphocytic Leukemia, Acute Myeloid Leukemia, Adrenocortical Carcinoma, Adult (Primary) Hepatocellular Cancer, Adult (Primary) Liver Cancer, Adult Acute Lymphocytic Leukemia, Adult Acute Myeloid Leukemia, Adult Hodgkin's Disease, Adult Hodgkin's Lymphoma, Adult Lymphocytic Leukemia, Adult Non-Hodgkin's Lymphoma, Adult Primary Liver Cancer, Adult Soft Tissue Sarcoma, AIDS-Related Lymphoma, AIDS-Related Malignancies, Anal Cancer, Astrocytoma, Bile Duct Cancer, Bladder Cancer, Bone Cancer, Brain Stem Glioma, Brain Tumors, Breast Cancer, Cancer of the Renal Pelvis and Ureter, Central Nervous System (Primary) Lymphoma, Central Nervous System Lymphoma, Cerebellar Astrocytoma, Cerebral Astrocytoma, Cervical Cancer, Childhood (Primary) Hepatocellular Cancer, Childhood (Primary) Liver Cancer, Childhood Acute Lymphoblastic Leukemia, Childhood Acute Myeloid Leukemia, Childhood Brain Stem Glioma, Childhood Cerebellar Astrocytoma, Childhood Cerebral Astrocytoma, Childhood Extracranial Germ Cell Tumors, Childhood Hodgkin's Disease, Childhood Hodgkin's Lymphoma, Childhood Hypothalamic and Visual Pathway Glioma, Childhood Lymphoblastic Leukemia, Childhood Medulloblastoma, Childhood Non-Hodgkin's Lymphoma, Childhood Pineal and Supratentorial Primitive Neuroectodermal Tumors, Childhood Primary Liver Cancer, Childhood Rhabdomyosarcoma, Childhood Soft Tissue Sarcoma, Childhood Visual Pathway and Hypothalamic Glioma, Chronic Lymphocytic Leukemia, Chronic Myelogenous Leukemia, Colon Cancer, Cutaneous T-Cell Lymphoma, Endocrine Pancreas Islet Cell Carcinoma, Endometrial Cancer, Ependymoma, Epithelial Cancer, Esophageal Cancer, Ewing's Sarcoma and Related Tumors, Exocrine Pancreatic Cancer, Extracranial Germ Cell Tumor, Extragonadal Germ Cell Tumor, Extrahepatic Bile Duct Cancer, Eye Cancer, Female Breast Cancer, Gaucher's Disease, Gallbladder Cancer, Gastric Cancer, Gastrointestinal Carcinoid Tumor, Gastrointestinal Tumors, Germ Cell Tumors, Gestational Trophoblastic Tumor, Hairy Cell Leukemia, Head and Neck Hodgkin's Disease, Hodgkin's Lymphoma, Hepatocellular Cancer, Cancer, Hypergammaglobulinemia, Hypopharyngeal Cancer, Intestinal Cancers, Intraocular Melanoma, Islet Cell Carcinoma, Islet Cell Pancreatic Cancer, Kaposi's Sarcoma, Kidney Cancer, Laryngeal Cancer, Lip and Oral Cavity Cancer, Liver Cancer, Lung Cancer, Lymphoproliferative Disorders, Macroglobulinemia, Male Breast Cancer, Malignant Mesothelioma, Malignant Thymoma, Medulloblastoma, Melanoma, Mesothelioma, Metastatic Occult Primary Squamous Neck Cancer, Metastatic Primary Squamous Neck Cancer, Metastatic Squamous Neck Cancer, Multiple Myeloma, Multiple Myeloma/Plasma Cell Neoplasm, Myelodysplastic Syndrome, Myelogenous

Leukemia, Myeloid Leukemia, Myeloproliferative Disorders, Nasal Cavity and Paranasal Sinus Cancer, Nasopharyngeal Cancer, Neuroblastoma, Non-Hodgkin's Lymphoma During Pregnancy, Nonmelanoma Skin Cancer, Non-Small Cell Lung Cancer, Occult Primary Metastatic Squamous Oropharyngeal Cancer, Osteo-/Malignant Fibrous Sarcoma. Neck Cancer, Osteosarcoma/Malignant Fibrous Histiocytoma, Osteosarcoma/Malignant Fibrous Histiocytoma of Bone, Ovarian Epithelial Cancer, Ovarian Germ Cell Tumor, Ovarian Low Malignant Potential Tumor, Pancreatic Cancer, Paraproteinemias, Purpura, Parathyroid Cancer, Penile Cancer, Pheochromocytoma, Pituitary Tumor, Plasma Cell Neoplasm/Multiple Myeloma, Primary Central Nervous System Lymphoma, Primary Liver Cancer, Prostate Cancer, Rectal Cancer, Renal Cell Cancer, Renal Pelvis and Ureter Cancer, Retinoblastoma, Rhabdomyosarcoma, Salivary Gland Cancer, Sarcoidosis Sarcomas, Sezary Syndrome, Skin Cancer, Small Cell Lung Cancer, Small Intestine Cancer, Soft Tissue Sarcoma, Squamous Neck Cancer, Stomach Cancer, Supratentorial Primitive Neuroectodermal and Pineal Tumors, T-Cell Lymphoma, Testicular Cancer, Thymoma, Thyroid Cancer, Transitional Cell Cancer of the Renal Pelvis and Ureter, Transitional Renal Pelvis and Ureter Cancer, Trophoblastic Tumors, Ureter and Renal Pelvis Cell Cancer, Urethral Cancer, Uterine Cancer, Uterine Sarcoma, Vaginal Cancer, Visual Pathway and Hypothalamic Glioma, Vulvar Cancer, Waldenstrom's Macroglobulinemia, Wilms' Tumor, and any hyperproliferative disease, besides neoplasia, located in an organ system listed above.

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In another preferred embodiment, polynucleotides or polypeptides, or agonists or antagonists of the present invention are used to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate premalignant conditions and to prevent progression to a neoplastic or malignant state, including but not limited to those disorders described above. Such uses are indicated in conditions known or suspected of preceding progression to neoplasia or cancer, in particular, where non-neoplastic cell growth consisting of hyperplasia, metaplasia, or most particularly, dysplasia has occurred (for review of such abnormal growth conditions, see Robbins and Angell, 1976, Basic Pathology, 2d Ed., W. B. Saunders Co., Philadelphia, pp. 68-79.)

Hyperplasia is a form of controlled cell proliferation, involving an increase in cell number in a tissue or organ, without significant alteration in structure or function. Hyperplastic disorders which can be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated with compositions of the invention (including polynucleotides, polypeptides, agonists or antagonists) include, but are not limited to, angiofollicular mediastinal lymph node hyperplasia, angiolymphoid hyperplasia with eosinophilia, atypical melanocytic hyperplasia, basal cell hyperplasia, benign giant lymph node hyperplasia, cementum hyperplasia, congenital adrenal hyperplasia, congenital sebaceous hyperplasia, cystic hyperplasia, cystic hyperplasia of the breast, denture hyperplasia, ductal hyperplasia, endometrial hyperplasia, fibromuscular hyperplasia, focal epithelial hyperplasia, gingival hyperplasia, inflammatory fibrous hyperplasia, inflammatory papillary hyperplasia, intravascular papillary endothelial hyperplasia, nodular hyperplasia of prostate,

nodular regenerative hyperplasia, pseudoepitheliomatous hyperplasia, senile sebaceous hyperplasia, and verrucous hyperplasia.

Metaplasia is a form of controlled cell growth in which one type of adult or fully differentiated cell substitutes for another type of adult cell. Metaplastic disorders which can be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated with compositions of the invention (including polynucleotides, polypeptides, agonists or antagonists) include, but are not limited to, agnogenic myeloid metaplasia, apocrine metaplasia, atypical metaplasia, autoparenchymatous metaplasia, connective tissue metaplasia, epithelial metaplasia, intestinal metaplasia, metaplastic anemia, metaplastic ossification, metaplastic polyps, myeloid metaplasia, primary myeloid metaplasia, secondary myeloid metaplasia, squamous metaplasia, squamous metaplasia of amnion, and symptomatic myeloid metaplasia.

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Dysplasia is frequently a forerunner of cancer, and is found mainly in the epithelia; it is the most disorderly form of non-neoplastic cell growth, involving a loss in individual cell uniformity and in the architectural orientation of cells. Dysplastic cells often have abnormally large, deeply stained nuclei, and exhibit pleomorphism. Dysplasia characteristically occurs where there exists chronic irritation or inflammation. Dysplastic disorders which can be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated with compositions of the invention (including polynucleotides, polypeptides, agonists or antagonists) include, but are not limited to, anhidrotic ectodermal dysplasia, anterofacial dysplasia, asphyxiating thoracic dysplasia, atriodigital dysplasia, bronchopulmonary dysplasia, cerebral dysplasia, cervical dysplasia, chondroectodermal dysplasia, cleidocranial dysplasia, congenital ectodermal dysplasia, craniodiaphysial dysplasia, craniocarpotarsal dysplasia, craniometaphysial dysplasia, dentin dysplasia, diaphysial dysplasia, ectodermal dysplasia, enamel dysplasia, encephalo-ophthalmic dysplasia, dysplasia epiphysialis hemimelia, dysplasia epiphysialis multiplex, dysplasia epiphysialis punctata, epithelial dysplasia, faciodigitogenital dysplasia, familial fibrous dysplasia of jaws, familial white folded dysplasia, fibromuscular dysplasia, fibrous dysplasia of bone, florid osseous dysplasia, hereditary renal-retinal dysplasia, hidrotic ectodermal dysplasia, hypohidrotic ectodermal dysplasia, lymphopenic thymic dysplasia, mammary dysplasia, mandibulofacial dysplasia, metaphysial dysplasia, Mondini dysplasia, monostotic fibrous dysplasia, mucoepithelial dysplasia, multiple epiphysial dysplasia, oculoauriculovertebral dysplasia, oculodentodigital dysplasia, oculovertebral dysplasia, odontogenic dysplasia, ophthalmomandibulomelic dysplasia, periapical cemental dysplasia, polyostotic fibrous dysplasia, pseudoachondroplastic spondyloepiphysial dysplasia, retinal dysplasia, septo-optic dysplasia, spondyloepiphysial dysplasia, and ventriculoradial dysplasia.

Additional pre-neoplastic disorders which can be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated with compositions of the invention (including polynucleotides, polypeptides, agonists or antagonists) include, but are not limited to, benign

dysproliferative disorders (e.g., benign tumors, fibrocystic conditions, tissue hypertrophy, intestinal polyps, colon polyps, and esophageal dysplasia), leukoplakia, keratoses, Bowen's disease, Farmer's Skin, solar cheilitis, and solar keratosis.

In another embodiment, a polypeptide of the invention, or polynucleotides, antibodies, agonists, or antagonists corresponding to that polypeptide, may be used to diagnose and/or prognosticate disorders associated with the tissue(s) in which the polypeptide of the invention is expressed, including one, two, three, four, five, or more tissues disclosed in Table 1B.2, column 5 (Tissue Distribution Library Code).

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In another embodiment, polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention conjugated to a toxin or a radioactive isotope, as described herein, may be used to treat cancers and neoplasms, including, but not limited to those described herein. In a further preferred embodiment, polynucleotides, polypeptides, antibodies, and/or agonists or antagonists of the present invention conjugated to a toxin or a radioactive isotope, as described herein, may be used to treat acute myelogenous leukemia.

Additionally, polynucleotides, polypeptides, and/or agonists or antagonists of the invention may affect apoptosis, and therefore, would be useful in treating a number of diseases associated with increased cell survival or the inhibition of apoptosis. For example, diseases associated with increased cell survival or the inhibition of apoptosis that could be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated by polynucleotides, polypeptides, and/or agonists or antagonists of the invention, include cancers (such as follicular lymphomas, carcinomas with p53 mutations, and hormone-dependent tumors, including, but not limited to colon cancer, cardiac tumors, pancreatic cancer, melanoma, retinoblastoma, glioblastoma, lung cancer, intestinal cancer, testicular cancer, stomach cancer, neuroblastoma, myxoma, myoma, lymphoma, endothelioma, osteoblastoma, osteoclastoma, osteosarcoma, chondrosarcoma, adenoma, breast cancer, prostate cancer, Kaposi's sarcoma and ovarian cancer); autoimmune disorders such as, multiple sclerosis, Sjogren's syndrome, Hashimoto's thyroiditis, biliary cirrhosis, Behcet's disease, Crohn's disease, polymyositis, systemic lupus erythematosus and immune-related glomerulonephritis and rheumatoid arthritis) and viral infections (such as herpes viruses, pox viruses and adenoviruses), inflammation, graft v. host disease, acute graft rejection, and chronic graft rejection.

In preferred embodiments, polynucleotides, polypeptides, and/or agonists or antagonists of the invention are used to inhibit growth, progression, and/or metastasis of cancers, in particular those listed above.

Additional diseases or conditions associated with increased cell survival that could be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated by polynucleotides, polypeptides, and/or agonists or antagonists of the invention, include, but are not limited to, progression, and/or metastases of malignancies and related disorders such as leukemia (including

acute leukemias (e.g., acute lymphocytic leukemia, acute myelocytic leukemia (including myeloblastic, promyelocytic, myelomonocytic, monocytic, and erythroleukemia)) and chronic leukemias (e.g., chronic myelocytic (granulocytic) leukemia and chronic lymphocytic leukemia)), polycythemia vera, lymphomas (e.g., Hodgkin's disease and non-Hodgkin's disease), multiple myeloma, Waldenstrom's macroglobulinemia, heavy chain disease, and solid tumors including, but not limited to, sarcomas and carcinomas such as fibrosarcoma, myxosarcoma, liposarcoma, chondrosarcoma. osteogenic sarcoma, chordoma, angiosarcoma, endotheliosarcoma, lymphangiosarcoma, lymphangioendotheliosarcoma, synovioma, mesothelioma, Ewing's tumor, leiomyosarcoma, rhabdomyosarcoma, colon carcinoma, pancreatic cancer, breast cancer, ovarian cancer, prostate cancer, squarnous cell carcinoma, basal cell carcinoma, adenocarcinoma, sweat gland carcinoma, sebaceous gland carcinoma, papillary carcinoma, papillary adenocarcinomas, cystadenocarcinoma, medullary carcinoma, bronchogenic carcinoma, renal cell carcinoma, hepatoma, bile duct carcinoma, choriocarcinoma, seminoma, embryonal carcinoma, Wilm's tumor, cervical cancer, testicular tumor, lung carcinoma, small cell lung carcinoma, bladder carcinoma, epithelial carcinoma, glioma, astrocytoma, medulloblastoma, craniopharyngioma, ependymoma, pinealoma, emangioblastoma, acoustic neuroma, oligodendroglioma, menangioma, melanoma, neuroblastoma, and retinoblastoma.

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Diseases associated with increased apoptosis that could be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated by polynucleotides, polypeptides, and/or agonists or antagonists of the invention, include AIDS; neurodegenerative disorders (such as Alzheimer's disease, Parkinson's disease, amyotrophic lateral sclerosis, retinitis pigmentosa, cerebellar degeneration and brain tumor or prior associated disease); autoimmune disorders (such as, multiple sclerosis, Sjogren's syndrome, Hashimoto's thyroiditis, biliary cirrhosis, Behcet's disease, Crohn's disease, polymyositis, systemic lupus erythematosus and immune-related glomerulonephritis and rheumatoid arthritis) myelodysplastic syndromes (such as aplastic anemia), graft v. host disease, ischemic injury (such as that caused by myocardial infarction, stroke and reperfusion injury), liver injury (e.g., hepatitis related liver injury, ischemia/reperfusion injury, cholestosis (bile duct injury) and liver cancer); toxin-induced liver disease (such as that caused by alcohol), septic shock, cachexia and anorexia.

Hyperproliferative diseases and/or disorders that could be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated by polynucleotides, polypeptides, and/or agonists or antagonists of the invention, include, but are not limited to, neoplasms located in the liver, abdomen, bone, breast, digestive system, pancreas, peritoneum, endocrine glands (adrenal, parathyroid, pituitary, testicles, ovary, thymus, thyroid), eye, head and neck, nervous system (central and peripheral), lymphatic system, pelvis, skin, soft tissue, spleen, thorax, and urogenital tract.

Similarly, other hyperproliferative disorders can also be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated by polynucleotides, polypeptides, and/or agonists or antagonists of the invention. Examples of such hyperproliferative disorders include, but are not limited to: hypergammaglobulinemia, lymphoproliferative disorders, paraproteinemias, purpura, sarcoidosis, Sezary Syndrome, Waldenstron's macroglobulinemia, Gaucher's Disease, histiocytosis, and any other hyperproliferative disease, besides neoplasia, located in an organ system listed above.

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Another preferred embodiment utilizes polynucleotides of the present invention to inhibit aberrant cellular division, by gene therapy using the present invention, and/or protein fusions or fragments thereof.

Thus, the present invention provides a method for treating cell proliferative disorders by inserting into an abnormally proliferating cell a polynucleotide of the present invention, wherein said polynucleotide represses said expression.

Another embodiment of the present invention provides a method of treating cellproliferative disorders in individuals comprising administration of one or more active gene copies of the present invention to an abnormally proliferating cell or cells. In a preferred embodiment, polynucleotides of the present invention is a DNA construct comprising a recombinant expression vector effective in expressing a DNA sequence encoding said polynucleotides. In another preferred embodiment of the present invention, the DNA construct encoding the poynucleotides of the present invention is inserted into cells to be treated utilizing a retrovirus, or more preferably an adenoviral vector (See G J. Nabel, et. al., PNAS 1999 96: 324-326, which is hereby incorporated by reference). In a most preferred embodiment, the viral vector is defective and will not transform non-proliferating cells, only proliferating cells. Moreover, in a preferred embodiment, the polynucleotides of the present invention inserted into proliferating cells either alone, or in combination with or fused to other polynucleotides, can then be modulated via an external stimulus (i.e. magnetic, specific small molecule, chemical, or drug administration, etc.), which acts upon the promoter upstream of said polynucleotides to induce expression of the encoded protein product. As such the beneficial therapeutic affect of the present invention may be expressly . modulated (i.e. to increase, decrease, or inhibit expression of the present invention) based upon said external stimulus.

Polynucleotides of the present invention may be useful in repressing expression of oncogenic genes or antigens. By "repressing expression of the oncogenic genes" is intended the suppression of the transcription of the gene, the degradation of the gene transcript (pre-message RNA), the inhibition of splicing, the destruction of the messenger RNA, the prevention of the post-translational modifications of the protein, the destruction of the protein, or the inhibition of the normal function of the protein.

For local administration to abnormally proliferating cells, polynucleotides of the present invention may be administered by any method known to those of skill in the art including, but not limited to transfection, electroporation, microinjection of cells, or in vehicles such as liposomes, lipofectin, or as naked polynucleotides, or any other method described throughout the specification. The polynucleotide of the present invention may be delivered by known gene delivery systems such as, but not limited to, retroviral vectors (Gilboa, J. Virology 44:845 (1982); Hocke, Nature 320:275 (1986); Wilson, et al., Proc. Natl. Acad. Sci. U.S.A. 85:3014), vaccinia virus system (Chakrabarty et al., Mol. Cell Biol. 5:3403 (1985) or other efficient DNA delivery systems (Yates et al., Nature 313:812 (1985)) known to those skilled in the art. These references are exemplary only and are hereby incorporated by reference. In order to specifically deliver or transfect cells which are abnormally proliferating and spare non-dividing cells, it is preferable to utilize a retrovirus, or adenoviral (as described in the art and elsewhere herein) delivery system known to those of skill in the art. Since host DNA replication is required for retroviral DNA to integrate and the retrovirus will be unable to self replicate due to the lack of the retrovirus genes needed for its life cycle. Utilizing such a retroviral delivery system for polynucleotides of the present invention will target said gene and constructs to abnormally proliferating cells and will spare the non-dividing normal cells.

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The polynucleotides of the present invention may be delivered directly to cell proliferative disorder/disease sites in internal organs, body cavities and the like by use of imaging devices used to guide an injecting needle directly to the disease site. The polynucleotides of the present invention may also be administered to disease sites at the time of surgical intervention.

By "cell proliferative disease" is meant any human or animal disease or disorder, affecting any one or any combination of organs, cavities, or body parts, which is characterized by single or multiple local abnormal proliferations of cells, groups of cells, or tissues, whether benign or malignant.

Any amount of the polynucleotides of the present invention may be administered as long as it has a biologically inhibiting effect on the proliferation of the treated cells. Moreover, it is possible to administer more than one of the polynucleotide of the present invention simultaneously to the same site. By "biologically inhibiting" is meant partial or total growth inhibition as well as decreases in the rate of proliferation or growth of the cells. The biologically inhibitory dose may be determined by assessing the effects of the polynucleotides of the present invention on target malignant or abnormally proliferating cell growth in tissue culture, tumor growth in animals and cell cultures, or any other method known to one of ordinary skill in the art.

The present invention is further directed to antibody-based therapies which involve administering of anti-polypeptides and anti-polynucleotide antibodies to a mammalian, preferably human, patient for treating one or more of the described disorders. Methods for producing anti-polypeptides and anti-polynucleotide antibodies polyclonal and monoclonal antibodies are

described in detail elsewhere herein. Such antibodies may be provided in pharmaceutically acceptable compositions as known in the art or as described herein.

A summary of the ways in which the antibodies of the present invention may be used therapeutically includes binding polynucleotides or polypeptides of the present invention locally or systemically in the body or by direct cytotoxicity of the antibody, e.g. as mediated by complement (CDC) or by effector cells (ADCC). Some of these approaches are described in more detail below. Armed with the teachings provided herein, one of ordinary skill in the art will know how to use the antibodies of the present invention for diagnosis, prognosis, monitoring, or therapeutic purposes without undue experimentation.

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In particular, the antibodies, fragments and derivatives of the present invention are useful for treating a subject having or developing cell proliferative and/or differentiation disorders as described herein. Such treatment comprises administering a single or multiple doses of the antibody, or a fragment, derivative, or a conjugate thereof.

The antibodies of this invention may be advantageously utilized in combination with other monoclonal or chimeric antibodies, or with lymphokines or hematopoietic growth factors, for example., which serve to increase the number or activity of effector cells which interact with the antibodies.

It is preferred to use high affinity and/or potent *in vivo* inhibiting and/or neutralizing antibodies against polypeptides or polynucleotides of the present invention, fragments or regions thereof, for both immunoassays directed to and therapy of disorders related to polynucleotides or polypeptides, including fragements thereof, of the present invention. Such antibodies, fragments, or regions, will preferably have an affinity for polynucleotides or polypeptides, including fragements thereof. Preferred binding affinities include those with a dissociation constant or Kd less than 5X10-6M, 10-6M, 5X10-7M, 10-7M, 5X10-8M, 10-8M, 5X10-9M, 10-9M, 5X10-10M, 10-10M, 5X10-11M, 10-11M, 5X10-12M, 10-12M, 5X10-13M, 10-13M, 5X10-14M, 10-14M, 5X10-15M, and 10-15M.

Moreover, polypeptides of the present invention are useful in inhibiting the angiogenesis of proliferative cells or tissues, either alone, as a protein fusion, or in combination with other polypeptides directly or indirectly, as described elsewhere herein. In a most preferred embodiment, said anti-angiogenesis effect may be achieved indirectly, for example, through the inhibition of hematopoietic, tumor-specific cells, such as tumor-associated macrophages (See Joseph IB, et al. J Natl Cancer Inst, 90(21):1648-53 (1998), which is hereby incorporated by reference). Antibodies directed to polypeptides or polynucleotides of the present invention may also result in inhibition of angiogenesis directly, or indirectly (See Witte L, et al., Cancer Metastasis Rev. 17(2):155-61 (1998), which is hereby incorporated by reference)).

Polypeptides, including protein fusions, of the present invention, or fragments thereof may be useful in inhibiting proliferative cells or tissues through the induction of apoptosis. Said

polypeptides may act either directly, or indirectly to induce apoptosis of proliferative cells and tissues, for example in the activation of a death-domain receptor, such as tumor necrosis factor (TNF) receptor-1, CD95 (Fas/APO-1), TNF-receptor-related apoptosis-mediated protein (TRAMP) and TNF-related apoptosis-inducing ligand (TRAIL) receptor-1 and -2 (See Schulze-Osthoff K, et.al., Eur J Biochem 254(3):439-59 (1998), which is hereby incorporated by reference). Moreover, in another preferred embodiment of the present invention, said polypeptides may induce apoptosis through other mechanisms, such as in the activation of other proteins which will activate apoptosis, or through stimulating the expression of said proteins, either alone or in combination with small molecule drugs or adjuviants, such as apoptonin, galectins, thioredoxins, anti-inflammatory proteins (See for example, Mutat Res 400(1-2):447-55 (1998), Med Hypotheses.50(5):423-33 (1998), Chem Biol Interact. Apr 24;111-112:23-34 (1998), J Mol Med.76(6):402-12 (1998), Int J Tissue React;20(1):3-15 (1998), which are all hereby incorporated by reference).

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Polypeptides, including protein fusions to, or fragments thereof, of the present invention are useful in inhibiting the metastasis of proliferative cells or tissues. Inhibition may occur as a direct result of administering polypeptides, or antibodies directed to said polypeptides as described elsewere herein, or indirectly, such as activating the expression of proteins known to inhibit metastasis, for example alpha 4 integrins, (See, e.g., Curr Top Microbiol Immunol 1998;231:125-41, which is hereby incorporated by reference). Such thereapeutic affects of the present invention may be achieved either alone, or in combination with small molecule drugs or adjuvants.

In another embodiment, the invention provides a method of delivering compositions containing the polypeptides of the invention (e.g., compositions containing polypeptides or polypeptide antibodes associated with heterologous polypeptides, heterologous nucleic acids, toxins, or prodrugs) to targeted cells expressing the polypeptide of the present invention. Polypeptides or polypeptide antibodes of the invention may be associated with with heterologous polypeptides, heterologous nucleic acids, toxins, or prodrugs via hydrophobic, hydrophilic, ionic and/or covalent interactions.

Polypeptides, protein fusions to, or fragments thereof, of the present invention are useful in enhancing the immunogenicity and/or antigenicity of proliferating cells or tissues, either directly, such as would occur if the polypeptides of the present invention 'vaccinated' the immune response to respond to proliferative antigens and immunogens, or indirectly, such as in activating the expression of proteins known to enhance the immune response (e.g. chemokines), to said antigens and immunogens.

# Cardiovascular Disorders

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Polynucleotides or polypeptides, or agonists or antagonists of the present invention, may be used to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate cardiovascular diseases and disorders, including, but not limited to, peripheral artery disease, such as limb ischemia.

Cardiovascular disorders include, but are not limited to, cardiovascular abnormalities, such as arterio-arterial fistula, arteriovenous fistula, cerebral arteriovenous malformations, congenital heart defects, pulmonary atresia, and Scimitar Syndrome. Congenital heart defects include, but are not limited to, aortic coarctation, cor triatriatum, coronary vessel anomalies, crisscross heart, dextrocardia, patent ductus arteriosus, Ebstein's anomaly, Eisenmenger complex, hypoplastic left heart syndrome, levocardia, tetralogy of fallot, transposition of great vessels, double outlet right ventricle, tricuspid atresia, persistent truncus arteriosus, and heart septal defects, such as aortopulmonary septal defect, endocardial cushion defects, Lutembacher's Syndrome, trilogy of Fallot, ventricular heart septal defects.

Cardiovascular disorders also include, but are not limited to, heart disease, such as arrhythmias, carcinoid heart disease, high cardiac output, low cardiac output, cardiac tamponade, endocarditis (including bacterial), heart aneurysm, cardiac arrest, congestive heart failure, congestive cardiomyopathy, paroxysmal dyspnea, cardiac edema, heart hypertrophy, congestive cardiomyopathy, left ventricular hypertrophy, right ventricular hypertrophy, post-infarction heart rupture, ventricular septal rupture, heart valve diseases, myocardial diseases, myocardial ischemia, pericardial effusion, pericarditis (including constrictive and tuberculous), pneumopericardium, postpericardiotomy syndrome, pulmonary heart disease, rheumatic heart disease, ventricular dysfunction, hyperemia, cardiovascular pregnancy complications, Scimitar Syndrome, cardiovascular syphilis, and cardiovascular tuberculosis.

Arrhythmias include, but are not limited to, sinus arrhythmia, atrial fibrillation, atrial flutter, bradycardia, extrasystole, Adams-Stokes Syndrome, bundle-branch block, sinoatrial block, long QT syndrome, parasystole, Lown-Ganong-Levine Syndrome, Mahaim-type pre-excitation syndrome, Wolff-Parkinson-White syndrome, sick sinus syndrome, tachycardias, and ventricular fibrillation. Tachycardias include paroxysmal tachycardia, supraventricular tachycardia, accelerated idioventricular rhythm, atrioventricular nodal reentry tachycardia, ectopic atrial tachycardia, ectopic junctional tachycardia, sinoatrial nodal reentry tachycardia, sinus tachycardia, Torsades de Pointes, and ventricular tachycardia.

Heart valve diseases include, but are not limited to, aortic valve insufficiency, aortic valve stenosis, hear murmurs, aortic valve prolapse, mitral valve prolapse, tricuspid valve prolapse, mitral valve insufficiency, mitral valve stenosis, pulmonary atresia, pulmonary valve insufficiency, pulmonary valve stenosis, tricuspid atresia, tricuspid valve insufficiency, and tricuspid valve stenosis.

Myocardial diseases include, but are not limited to, alcoholic cardiomyopathy, congestive cardiomyopathy, hypertrophic cardiomyopathy, aortic subvalvular stenosis, pulmonary subvalvular stenosis, restrictive cardiomyopathy, Chagas cardiomyopathy, endocardial fibroelastosis, endomyocardial fibrosis, Kearns Syndrome, myocardial reperfusion injury, and myocarditis.

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Myocardial ischemias include, but are not limited to, coronary disease, such as angina pectoris, coronary aneurysm, coronary arteriosclerosis, coronary thrombosis, coronary vasospasm, myocardial infarction and myocardial stunning.

Cardiovascular diseases also include vascular diseases such as aneurysms, angiodysplasia, angiomatosis, bacillary angiomatosis, Hippel-Lindau Disease, Klippel-Trenaunay-Weber Syndrome, Sturge-Weber Syndrome, angioneurotic edema, aortic diseases, Takayasu's Arteritis, aortitis, Leriche's Syndrome, arterial occlusive diseases, arteritis, enarteritis, polyarteritis nodosa, cerebrovascular disorders, diabetic angiopathies, diabetic retinopathy, embolisms, thrombosis, erythromelalgia, hemorrhoids, hepatic veno-occlusive disease, hypertension, hypotension, ischemia, peripheral vascular diseases, phlebitis, pulmonary veno-occlusive disease, Raynaud's disease, CREST syndrome, retinal vein occlusion, Scimitar syndrome, superior vena cava syndrome, telangiectasia, atacia telangiectasia, hereditary hemorrhagic telangiectasia, varicocele, varicose veins, varicose ulcer, vasculitis, and venous insufficiency.

Aneurysms include, but are not limited to, dissecting aneurysms, false aneurysms, infected aneurysms, ruptured aneurysms, aortic aneurysms, cerebral aneurysms, coronary aneurysms, heart aneurysms, and iliac aneurysms.

Arterial occlusive diseases include, but are not limited to, arteriosclerosis, intermittent claudication, carotid stenosis, fibromuscular dysplasias, mesenteric vascular occlusion, Moyamoya disease, renal artery obstruction, retinal artery occlusion, and thromboangiitis obliterans.

Cerebrovascular disorders include, but are not limited to, carotid artery diseases, cerebral amyloid angiopathy, cerebral aneurysm, cerebral anoxia, cerebral arteriosclerosis, cerebral arteriovenous malformation, cerebral artery diseases, cerebral embolism and thrombosis, carotid artery thrombosis, sinus thrombosis, Wallenberg's syndrome, cerebral hemorrhage, epidural hematoma, subdural hematoma, subaraxhnoid hemorrhage, cerebral infarction, cerebral ischemia (including transient), subclavian steal syndrome, periventricular leukomalacia, vascular headache, cluster headache, migraine, and vertebrobasilar insufficiency.

Embolisms include, but are not limited to, air embolisms, amniotic fluid embolisms, cholesterol embolisms, blue toe syndrome, fat embolisms, pulmonary embolisms, and thromoboembolisms. Thrombosis include, but are not limited to, coronary thrombosis, hepatic vein thrombosis, retinal vein occlusion, carotid artery thrombosis, sinus thrombosis, Wallenberg's syndrome, and thrombophlebitis.

Ischemic disorders include, but are not limited to, cerebral ischemia, ischemic colitis, compartment syndromes, anterior compartment syndrome, myocardial ischemia, reperfusion injuries, and peripheral limb ischemia. Vasculitis includes, but is not limited to, aortitis, arteritis, Behcet's Syndrome, Churg-Strauss Syndrome, mucocutaneous lymph node syndrome, thromboangiitis obliterans, hypersensitivity vasculitis, Schoenlein-Henoch purpura, allergic cutaneous vasculitis, and Wegener's granulomatosis.

Polypeptides may be administered using any method known in the art, including, but not limited to, direct needle injection at the delivery site, intravenous injection, topical administration, catheter infusion, biolistic injectors, particle accelerators, gelfoam sponge depots, other commercially available depot materials, osmotic pumps, oral or suppositorial solid pharmaceutical formulations, decanting or topical applications during surgery, aerosol delivery. Such methods are known in the art. Polypeptides may be administered as part of a Therapeutic, described in more detail below. Methods of delivering polynucleotides are described in more detail herein.

## 15 Diseases at the Cellular Level

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Diseases associated with increased cell survival or the inhibition of apoptosis that could be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated using polynucleotides or polypeptides, as well as antagonists or agonists of the present invention, include cancers (such as follicular lymphomas, carcinomas with p53 mutations, and hormone-dependent tumors, including, but not limited to colon cancer, cardiac tumors, pancreatic cancer, melanoma, retinoblastoma, glioblastoma, lung cancer, intestinal cancer, testicular cancer, stomach cancer, neuroblastoma, myxoma, myoma, lymphoma, endothelioma, osteoblastoma, osteoclastoma, osteosarcoma, chondrosarcoma, adenoma, breast cancer, prostate cancer, Kaposi's sarcoma and ovarian cancer); autoimmune disorders (such as, multiple sclerosis, Sjogren's syndrome, Hashimoto's thyroiditis, biliary cirrhosis, Behcet's disease, Crohn's disease, polymyositis, systemic lupus erythematosus and immune-related glomerulonephritis and rheumatoid arthritis) and viral infections (such as herpes viruses, pox viruses and adenoviruses), inflammation, graft v. host disease, acute graft rejection, and chronic graft rejection.

In preferred embodiments, polynucleotides, polypeptides, and/or antagonists of the invention are used to inhibit growth, progression, and/or metasis of cancers, in particular those listed above.

Additional diseases or conditions associated with increased cell survival that could be treated or detected by polynucleotides or polypeptides, or agonists or antagonists of the present invention include, but are not limited to, progression, and/or metastases of malignancies and related disorders such as leukemia (including acute leukemias (e.g., acute lymphocytic leukemia, acute myelocytic leukemia (including myeloblastic, promyelocytic, myelomonocytic, monocytic, and erythroleukemia)) and chronic leukemias (e.g., chronic myelocytic (granulocytic) leukemia

and chronic lymphocytic leukemia)), polycythemia vera, lymphomas (e.g., Hodgkin's disease and non-Hodgkin's disease), multiple myeloma, Waldenstrom's macroglobulinemia, heavy chain disease, and solid tumors including, but not limited to, sarcomas and carcinomas such as fibrosarcoma, myxosarcoma, liposarcoma, chondrosarcoma, osteogenic sarcoma, chordoma, lymphangioendotheliosarcoma, angiosarcoma, endotheliosarcoma, lymphangiosarcoma, synovioma, mesothelioma, Ewing's tumor, leiomyosarcoma, rhabdomyosarcoma, colon carcinoma, pancreatic cancer, breast cancer, ovarian cancer, prostate cancer, squamous cell carcinoma, basal cell carcinoma, adenocarcinoma, sweat gland carcinoma, sebaceous gland carcinoma, papillary carcinoma, papillary adenocarcinomas, cystadenocarcinoma, medullary carcinoma, bronchogenic carcinoma, renal cell carcinoma, hepatoma, bile duct carcinoma, choriocarcinoma, seminoma, embryonal carcinoma, Wilm's tumor, cervical cancer, testicular tumor, lung carcinoma, small cell lung carcinoma, bladder carcinoma, epithelial carcinoma, craniopharyngioma, ependymoma, pinealoma. glioma, astrocytoma, medulloblastoma, oligodendroglioma, menangioma, melanoma, hemangioblastoma, acoustic neuroma, neuroblastoma, and retinoblastoma.

Diseases associated with increased apoptosis that could be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated using polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, include, but are not limited to, AIDS; neurodegenerative disorders (such as Alzheimer's disease, Parkinson's disease, Amyotrophic lateral sclerosis, Retinitis pigmentosa, Cerebellar degeneration and brain tumor or prior associated disease); autoimmune disorders (such as, multiple sclerosis, Sjogren's syndrome, Hashimoto's thyroiditis, biliary cirrhosis, Behcet's disease, Crohn's disease, polymyositis, systemic lupus erythematosus and immune-related glomerulonephritis and rheumatoid arthritis) myelodysplastic syndromes (such as aplastic anemia), graft v. host disease, ischemic injury (such as that caused by myocardial infarction, stroke and reperfusion injury), liver injury (e.g., hepatitis related liver injury, ischemia/reperfusion injury, cholestosis (bile duct injury) and liver cancer); toxin-induced liver disease (such as that caused by alcohol), septic shock, cachexia and anorexia.

## Wound Healing and Epithelial Cell Proliferation

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In accordance with yet a further aspect of the present invention, there is provided a process for utilizing polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, for therapeutic purposes, for example, to stimulate epithelial cell proliferation and basal keratinocytes for the purpose of wound healing, and to stimulate hair follicle production and healing of dermal wounds. Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, may be clinically useful in stimulating wound healing including surgical wounds, excisional wounds, deep wounds involving damage of the dermis and epidermis, eye tissue wounds, dental tissue wounds, oral cavity wounds, diabetic ulcers, dermal ulcers, cubitus

ulcers, arterial ulcers, venous stasis ulcers, burns resulting from heat exposure or chemicals, and other abnormal wound healing conditions such as uremia, malnutrition, vitamin deficiencies and complications associated with systemic treatment with steroids, radiation therapy and antineoplastic drugs and antimetabolites. Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could be used to promote dermal reestablishment subsequent to dermal loss

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Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could be used to increase the adherence of skin grafts to a wound bed and to stimulate re-epithelialization from the wound bed. The following are types of grafts that polynucleotides or polypeptides, agonists or antagonists of the present invention, could be used to increase adherence to a wound bed: autografts, artificial skin, allografts, autodermic graft, autoepdermic grafts, avacular grafts, Blair-Brown grafts, bone graft, brephoplastic grafts, cutis graft, delayed graft, dermic graft, epidermic graft, fascia graft, full thickness graft, heterologous graft, xenograft, homologous graft, hyperplastic graft, lamellar graft, mesh graft, mucosal graft, Ollier-Thiersch graft, omenpal graft, patch graft, pedicle graft, penetrating graft, split skin graft, thick split graft. Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, can be used to promote skin strength and to improve the appearance of aged skin.

It is believed that polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, will also produce changes in hepatocyte proliferation, and epithelial cell proliferation in the lung, breast, pancreas, stomach, small intestine, and large intestine. Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could promote proliferation of epithelial cells such as sebocytes, hair follicles, hepatocytes, type II pneumocytes, mucin-producing goblet cells, and other epithelial cells and their progenitors contained within the skin, lung, liver, and gastrointestinal tract. Polynucleotides or polypeptides, agonists or antagonists of the present invention, may promote proliferation of endothelial cells, keratinocytes, and basal keratinocytes.

Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could also be used to reduce the side effects of gut toxicity that result from radiation, chemotherapy treatments or viral infections. Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, may have a cytoprotective effect on the small intestine mucosa. Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, may also stimulate healing of mucositis (mouth ulcers) that result from chemotherapy and viral infections.

Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could further be used in full regeneration of skin in full and partial thickness skin defects, including burns, (i.e., repopulation of hair follicles, sweat glands, and sebaceous glands), treatment of other skin defects such as psoriasis. Polynucleotides or polypeptides, as well as

agonists or antagonists of the present invention, could be used to treat epidermolysis bullosa, a defect in adherence of the epidermis to the underlying dermis which results in frequent, open and painful blisters by accelerating reepithelialization of these lesions. Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could also be used to treat gastric and doudenal ulcers and help heal by scar formation of the mucosal lining and regeneration of glandular mucosa and duodenal mucosal lining more rapidly. Inflammatory bowel diseases, such as Crohn's disease and ulcerative colitis, are diseases which result in destruction of the mucosal surface of the small or large intestine, respectively. Thus, polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could be used to promote the resurfacing of the mucosal surface to aid more rapid healing and to prevent progression of inflammatory bowel disease. Treatment with polynucleotides or polypeptides, agonists or antagonists of the present invention, is expected to have a significant effect on the production of mucus throughout the gastrointestinal tract and could be used to protect the intestinal mucosa from injurious substances that are ingested or following surgery. Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could be used to treat diseases associate with the under expression.

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Moreover, polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could be used to prevent and heal damage to the lungs due to various pathological states. Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, which could stimulate proliferation and differentiation and promote the repair of alveoli and brochiolar epithelium to prevent or treat acute or chronic lung damage. For example, emphysema, which results in the progressive loss of aveoli, and inhalation injuries, i.e., resulting from smoke inhalation and burns, that cause necrosis of the bronchiolar epithelium and alveoli could be effectively treated using polynucleotides or polypeptides, agonists or antagonists of the present invention. Also, polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could be used to stimulate the proliferation of and differentiation of type II pneumocytes, which may help treat or prevent disease such as hyaline membrane diseases, such as infant respiratory distress syndrome and bronchopulmonary displasia, in premature infants.

Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could stimulate the proliferation and differentiation of hepatocytes and, thus, could be used to alleviate or treat liver diseases and pathologies such as fulminant liver failure caused by cirrhosis, liver damage caused by viral hepatitis and toxic substances (i.e., acetaminophen, carbon tetraholoride and other hepatotoxins known in the art).

In addition, polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could be used treat or prevent the onset of diabetes mellitus. In patients with newly diagnosed Types I and II diabetes, where some islet cell function remains, polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could be used to

maintain the islet function so as to alleviate, delay or prevent permanent manifestation of the disease. Also, polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, could be used as an auxiliary in islet cell transplantation to improve or promote islet cell function.

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#### Infectious Disease

Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention can be used to treat or detect infectious agents. For example, by increasing the immune response, particularly increasing the proliferation and differentiation of B and/or T cells, infectious diseases may be treated. The immune response may be increased by either enhancing an existing immune response, or by initiating a new immune response. Alternatively, polynucleotides or polypeptides; as well as agonists or antagonists of the present invention may also directly inhibit the infectious agent, without necessarily eliciting an immune response.

Viruses are one example of an infectious agent that can cause disease or symptoms that can be treated or detected by a polynucleotide or polypeptide and/or agonist or antagonist of the present invention. Examples of viruses, include, but are not limited to Examples of viruses, include, but are not limited to the following DNA and RNA viruses and viral families: Arbovirus, Adenoviridae, Arenaviridae, Arterivirus, Birnaviridae, Bunyaviridae, Caliciviridae, Circoviridae, Coronaviridae, Dengue, EBV, HIV, Flaviviridae, Hepadnaviridae (Hepatitis), Herpesviridae (such as, Cytomegalovirus, Herpes Simplex, Herpes Zoster), Mononegavirus (e.g., Paramyxoviridae, Morbillivirus, Rhabdoviridae), Orthomyxoviridae (e.g., Influenza A, Influenza B, and parainfluenza), Papiloma virus, Papovaviridae, Parvoviridae, Picornaviridae, Poxviridae (such as Smallpox or Vaccinia), Reoviridae (e.g., Rotavirus), Retroviridae (HTLV-I, HTLV-II, Lentivirus), and Togaviridae (e.g., Rubivirus). Viruses falling within these families can cause a variety of diseases or symptoms, including, but not limited to: arthritis, bronchiollitis, respiratory syncytial virus, encephalitis, eye infections (e.g., conjunctivitis, keratitis), chronic fatigue syndrome, hepatitis (A, B, C, E, Chronic Active, Delta), Japanese B encephalitis, Junin, Chikungunya, Rift Valley fever, yellow fever, meningitis, opportunistic infections (e.g., AIDS), pneumonia, Burkitt's Lymphoma, chickenpox, hemorrhagic fever, Measles, Mumps, Parainfluenza, Rabies, the common cold, Polio, leukemia, Rubella, sexually transmitted diseases, skin diseases (e.g., Kaposi's, warts), and viremia. polynucleotides or polypeptides, or agonists or antagonists of the invention, can be used to treat or detect any of these symptoms or diseases. In specific embodiments, polynucleotides, polypeptides, or agonists or antagonists of the invention are used to treat: meningitis, Dengue, EBV, and/or hepatitis (e.g., hepatitis B). In an additional specific embodiment polynucleotides, polypeptides, or agonists or antagonists of the invention are used to treat patients nonresponsive to one or more other commercially available hepatitis vaccines. In a

further specific embodiment polynucleotides, polypeptides, or agonists or antagonists of the invention are used to treat AIDS.

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Similarly, bacterial and fungal agents that can cause disease or symptoms and that can be treated or detected by a polynucleotide or polypeptide and/or agonist or antagonist of the present invention include, but not limited to, the following Gram-Negative and Gram-positive bacteria, bacterial families, and fungi: Actinomyces (e.g., Norcardia), Acinetobacter, Cryptococcus neoformans, Aspergillus, Bacillaceae (e.g., Bacillus anthrasis), Bacteroides (e.g., Bacteroides fragilis), Blastomycosis, Bordetella, Borrelia (e.g., Borrelia burgdorferi), Brucella, Candidia, Campylobacter, Chlamydia, Clostridium (e.g., Clostridium botulinum, Clostridium dificile, Clostridium tetani), Coccidioides, Corynebacterium (e.g., Clostridium perfringens, Corynebacterium diptheriae), Cryptococcus, Dermatocycoses, E. coli (e.g., Enterotoxigenic E. coli and Enterohemorrhagic E. coli), Enterobacter (e.g. Enterobacter aerogenes), Enterobacteriaceae (Klebsiella, Salmonella (e.g., Salmonella typhi, Salmonella enteritidis, Salmonella typhi), Serratia, Yersinia, Shigella), Erysipelothrix, Haemophilus (e.g., Haemophilus influenza type B), Helicobacter, Legionella (e.g., Legionella pneumophila), Leptospira, Listeria (e.g., Listeria monocytogenes), Mycoplasma, Mycobacterium (e.g., Mycobacterium leprae and Mycobacterium tuberculosis), Vibrio (e.g., Vibrio cholerae), Neisseriaceae (e.g., Neisseria gonorrhea, Neisseria meningitidis), Pasteurellacea, Proteus, Pseudomonas (e.g., Pseudomonas aeruginosa), Rickettsiaceae, Spirochetes (e.g., Treponema spp., Leptospira spp., Borrelia spp.), Shigella spp., Staphylococcus (e.g., Staphylococcus aureus), Meningiococcus, Pneumococcus and Streptococcus (e.g., Streptococcus pneumoniae and Groups A, B, and C Streptococci), and Ureaplasmas. These bacterial, parasitic, and fungal families can cause diseases or symptoms, including, but not limited to: antibiotic-resistant infections, bacteremia, endocarditis, septicemia, eye infections (e.g., conjunctivitis), uveitis, tuberculosis, gingivitis, bacterial diarrhea, opportunistic infections (e.g., AIDS related infections), paronychia, prosthesis-related infections, dental caries, Reiter's Disease, respiratory tract infections, such as Whooping Cough or Empyema, sepsis, Lyme Disease, Cat-Scratch Disease, dysentery, paratyphoid fever, food poisoning, Legionella disease, chronic and acute inflammation, erythema, yeast infections, typhoid, pneumonia, gonorrhea, meningitis (e.g., mengitis types A and B), chlamydia, syphillis, diphtheria, leprosy, brucellosis, peptic ulcers, anthrax, spontaneous abortions, birth defects, pneumonia, lung infections, ear infections, deafness, blindness, lethargy, malaise, vomiting, chronic diarrhea, Crohn's disease, colitis, vaginosis, sterility, pelvic inflammatory diseases, candidiasis, paratuberculosis, tuberculosis, lupus, botulism, gangrene, tetanus, impetigo, Rheumatic Fever, Scarlet Fever, sexually transmitted diseases, skin diseases (e.g., cellulitis, dermatocycoses), toxemia, urinary tract infections, wound infections, noscomial infections. Polynucleotides or polypeptides, agonists or antagonists of the invention, can be used to treat or detect any of these symptoms or diseases. In specific embodiments, polynucleotides, polypeptides, agonists or

antagonists of the invention are used to treat: tetanus, diptheria, botulism, and/or meningitis type B.

Moreover, parasitic agents causing disease or symptoms that can be detected, prevented, diagnosed, prognosticated, treated, and/or ameliorated by a polynucleotide or polypeptide and/or agonist or antagonist of the present invention include, but not limited to, the following families or class: Amebiasis, Babesiosis, Coccidiosis, Cryptosporidiosis, Dientamoebiasis, Dourine, Ectoparasitic, Giardias, Helminthiasis, Leishmaniasis, Schistisoma, Theileriasis, Toxoplasmosis, Trypanosomiasis, and Trichomonas and Sporozoans (e.g., Plasmodium virax, Plasmodium falciparium, Plasmodium malariae and Plasmodium ovale). These parasites can cause a variety of diseases or symptoms, including, but not limited to: Scabies, Trombiculiasis, eye infections, intestinal disease (e.g., dysentery, giardiasis), liver disease, lung disease, opportunistic infections (e.g., AIDS related), malaria, pregnancy complications, and toxoplasmosis. polynucleotides or polypeptides, or agonists or antagonists of the invention, can be used to detect, prevent, diagnose, prognosticate, treat, and/or ameliorate any of these symptoms or diseases. In specific embodiments, polynucleotides, polypeptides, or agonists or antagonists of the invention are used to detect, prevent, diagnose, treat, and/or ameliorate malaria.

Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention of the present invention could either be by administering an effective amount of a polypeptide to the patient, or by removing cells from the patient, supplying the cells with a polynucleotide of the present invention, and returning the engineered cells to the patient (ex vivo therapy). Moreover, the polypeptide or polynucleotide of the present invention can be used as an antigen in a vaccine to raise an immune response against infectious disease.

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#### Regeneration

Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention can be used to differentiate, proliferate, and attract cells, leading to the regeneration of tissues. (See, Science 276:59-87 (1997)). The regeneration of tissues could be used to repair, replace, or protect tissue damaged by congenital defects, trauma (wounds, burns, incisions, or ulcers), age, disease (e.g. osteoporosis, osteocarthritis, periodontal disease, liver failure), surgery, including cosmetic plastic surgery, fibrosis, reperfusion injury, or systemic cytokine damage.

Tissues that could be regenerated using the present invention include organs (e.g., pancreas, liver, intestine, kidney, skin, endothelium), muscle (smooth, skeletal or cardiac), vasculature (including vascular and lymphatics), nervous, hematopoietic, and skeletal (bone, cartilage, tendon, and ligament) tissue. Preferably, regeneration occurs without or decreased scarring. Regeneration also may include angiogenesis.

Moreover, polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, may increase regeneration of tissues difficult to heal. For example, increased tendon/ligament regeneration would quicken recovery time after damage. Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention could also be used prophylactically in an effort to avoid damage. Specific diseases that could be treated include of tendinitis, carpal tunnel syndrome, and other tendon or ligament defects. A further example of tissue regeneration of non-healing wounds includes pressure ulcers, ulcers associated with vascular insufficiency, surgical, and traumatic wounds.

Similarly, nerve and brain tissue could also be regenerated by using polynucleotides or polypeptides, as well as agonists or antagonists of the present invention, to proliferate and differentiate nerve cells. Diseases that could be treated using this method include central and peripheral nervous system diseases, neuropathies, or mechanical and traumatic disorders (e.g., spinal cord disorders, head trauma, cerebrovascular disease, and stoke). Specifically, diseases associated with peripheral nerve injuries, peripheral neuropathy (e.g., resulting from chemotherapy or other medical therapies), localized neuropathies, and central nervous system diseases (e.g., Alzheimer's disease, Parkinson's disease, Huntington's disease, amyotrophic lateral sclerosis, and Shy-Drager syndrome), could all be treated using the polynucleotides or polypeptides, as well as agonists or antagonists of the present invention.

#### **Chemotaxis**

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Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention may have chemotaxis activity. A chemotaxic molecule attracts or mobilizes cells (e.g., monocytes, fibroblasts, neutrophils, T-cells, mast cells, eosinophils, epithelial and/or endothelial cells) to a particular site in the body, such as inflammation, infection, or site of hyperproliferation. The mobilized cells can then fight off and/or heal the particular trauma or abnormality.

Polynucleotides or polypeptides, as well as agonists or antagonists of the present invention may increase chemotaxic activity of particular cells. These chemotactic molecules can then be used to treat inflammation, infection, hyperproliferative disorders, or any immune system disorder by increasing the number of cells targeted to a particular location in the body. For example, chemotaxic molecules can be used to treat wounds and other trauma to tissues by attracting immune cells to the injured location. Chemotactic molecules of the present invention can also attract fibroblasts, which can be used to treat wounds.

It is also contemplated that polynucleotides or polypeptides, as well as agonists or antagonists of the present invention may inhibit chemotactic activity. These molecules could also be used to treat disorders. Thus, polynucleotides or polypeptides, as well as agonists or antagonists of the present invention could be used as an inhibitor of chemotaxis.

### **Binding Activity**

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A polypeptide of the present invention may be used to screen for molecules that bind to the polypeptide or for molecules to which the polypeptide binds. The binding of the polypeptide and the molecule may activate (agonist), increase, inhibit (antagonist), or decrease activity of the polypeptide or the molecule bound. Examples of such molecules include antibodies, oligonucleotides, proteins (e.g., receptors), or small molecules.

Preferably, the molecule is closely related to the natural ligand of the polypeptide, e.g., a fragment of the ligand, or a natural substrate, a ligand, a structural or functional mimetic. (See, Coligan et al., Current Protocols in Immunology 1(2):Chapter 5 (1991)). Similarly, the molecule can be closely related to the natural receptor to which the polypeptide binds, or at least, a fragment of the receptor capable of being bound by the polypeptide (e.g., active site). In either case, the molecule can be rationally designed using known techniques.

Preferably, the screening for these molecules involves producing appropriate cells which express the polypeptide. Preferred cells include cells from mammals, yeast, Drosophila, or *E. coli*. Cells expressing the polypeptide (or cell membrane containing the expressed polypeptide) are then preferably contacted with a test compound potentially containing the molecule to observe binding, stimulation, or inhibition of activity of either the polypeptide or the molecule.

The assay may simply test binding of a candidate compound to the polypeptide, wherein binding is detected by a label, or in an assay involving competition with a labeled competitor. Further, the assay may test whether the candidate compound results in a signal generated by binding to the polypeptide.

Alternatively, the assay can be carried out using cell-free preparations, polypeptide/molecule affixed to a solid support, chemical libraries, or natural product mixtures. The assay may also simply comprise the steps of mixing a candidate compound with a solution containing a polypeptide, measuring polypeptide/molecule activity or binding, and comparing the polypeptide/molecule activity or binding to a standard.

Preferably, an ELISA assay can measure polypeptide level or activity in a sample (e.g., biological sample) using a monoclonal or polyclonal antibody. The antibody can measure polypeptide level or activity by either binding, directly or indirectly, to the polypeptide or by competing with the polypeptide for a substrate.

Additionally, the receptor to which the polypeptide of the present invention binds can be identified by numerous methods known to those of skill in the art, for example, ligand panning and FACS sorting (Coligan, et al., Current Protocols in Immun., 1(2), Chapter 5, (1991)). For example, expression cloning is employed wherein polyadenylated RNA is prepared from a cell responsive to the polypeptides, for example, NIH3T3 cells which are known to contain multiple receptors for the FGF family proteins, and SC-3 cells, and a cDNA library created from this RNA is divided into pools and used to transfect COS cells or other cells that are not responsive to the

polypeptides. Transfected cells which are grown on glass slides are exposed to the polypeptide of the present invention, after they have been labeled. The polypeptides can be labeled by a variety of means including iodination or inclusion of a recognition site for a site-specific protein kinase.

Following fixation and incubation, the slides are subjected to auto-radiographic analysis. Positive pools are identified and sub-pools are prepared and re-transfected using an iterative sub-pooling and re-screening process, eventually yielding a single clones that encodes the putative receptor.

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As an alternative approach for receptor identification, the labeled polypeptides can be photoaffinity linked with cell membrane or extract preparations that express the receptor molecule. Cross-linked material is resolved by PAGE analysis and exposed to X-ray film. The labeled complex containing the receptors of the polypeptides can be excised, resolved into peptide fragments, and subjected to protein microsequencing. The amino acid sequence obtained from microsequencing would be used to design a set of degenerate oligonucleotide probes to screen a cDNA library to identify the genes encoding the putative receptors.

Moreover, the techniques of gene-shuffling, motif-shuffling, exon-shuffling, and/or codon-shuffling (collectively referred to as "DNA shuffling") may be employed to modulate the activities of the polypeptide of the present invention thereby effectively generating agonists and antagonists of the polypeptide of the present invention. See generally, U.S. Patent Nos. 5,605,793, 5,811,238, 5,830,721, 5,834,252, and 5,837,458, and Patten, P. A., et al., Curr. Opinion Biotechnol. 8:724-33 (1997); Harayama, S. Trends Biotechnol. 16(2):76-82 (1998); Hansson, L. O., et al., J. Mol. Biol. 287:265-76 (1999); and Lorenzo, M. M. and Blasco, R. Biotechniques 24(2):308-13 (1998); each of these patents and publications are hereby incorporated by reference). In one embodiment, alteration of polynucleotides and corresponding polypeptides may be achieved by DNA shuffling. DNA shuffling involves the assembly of two or more DNA segments into a desired molecule by homologous, or site-specific, recombination. In another embodiment, polynucleotides and corresponding polypeptides may be altered by being subjected to random mutagenesis by error-prone PCR, random nucleotide insertion or other methods prior to recombination. In another embodiment, one or more components, motifs, sections, parts, domains, fragments, etc., of the polypeptide of the present invention may be recombined with one or more components, motifs, sections, parts, domains, fragments, etc. of one or more heterologous molecules. In preferred embodiments, the heterologous molecules are family members. In further preferred embodiments, the heterologous molecule is a growth factor such as, for example, platelet-derived growth factor (PDGF), insulin-like growth factor (IGF-I), transforming growth factor (TGF)-alpha, epidermal growth factor (EGF), fibroblast growth factor (FGF), TGF-beta, bone morphogenetic protein (BMP)-2, BMP-4, BMP-5, BMP-6, BMP-7, activins A and B, decapentaplegic(dpp), 60A, OP-2, dorsalin, growth differentiation factors (GDFs), nodal, MIS,

inhibin-alpha, TGF-beta1, TGF-beta2, TGF-beta3, TGF-beta5, and glial-derived neurotrophic factor (GDNF).

Other preferred fragments are biologically active fragments of the polypeptide of the present invention. Biologically active fragments are those exhibiting activity similar, but not necessarily identical, to an activity of the polypeptide of the present invention. The biological activity of the fragments may include an improved desired activity, or a decreased undesirable activity.

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Additionally, this invention provides a method of screening compounds to identify those which modulate the action of the polypeptide of the present invention. An example of such an assay comprises combining a mammalian fibroblast cell, a the polypeptide of the present invention, the compound to be screened and ³[H] thymidine under cell culture conditions where the fibroblast cell would normally proliferate. A control assay may be performed in the absence of the compound to be screened and compared to the amount of fibroblast proliferation in the presence of the compound to determine if the compound stimulates proliferation by determining the uptake of ³[H] thymidine in each case. The amount of fibroblast cell proliferation is measured by liquid scintillation chromatography which measures the incorporation of ³[H] thymidine. Both agonist and antagonist compounds may be identified by this procedure.

In another method, a mammalian cell or membrane preparation expressing a receptor for a polypeptide of the present invention is incubated with a labeled polypeptide of the present invention in the presence of the compound. The ability of the compound to enhance or block this interaction could then be measured. Alternatively, the response of a known second messenger system following interaction of a compound to be screened and the receptor is measured and the ability of the compound to bind to the receptor and elicit a second messenger response is measured to determine if the compound is a potential agonist or antagonist. Such second messenger systems include but are not limited to, cAMP guanylate cyclase, ion channels or phosphoinositide hydrolysis.

All of these above assays can be used as diagnostic or prognostic markers. The molecules discovered using these assays can be used to treat disease or to bring about a particular result in a patient (e.g., blood vessel growth) by activating or inhibiting the polypeptide/molecule. Moreover, the assays can discover agents which may inhibit or enhance the production of the polypeptides of the invention from suitably manipulated cells or tissues.

Therefore, the invention includes a method of identifying compounds which bind to a polypeptide of the invention comprising the steps of: (a) incubating a candidate binding compound with a polypeptide of the present invention; and (b) determining if binding has occurred. Moreover, the invention includes a method of identifying agonists/antagonists comprising the steps of: (a) incubating a candidate compound with a polypeptide of the present

invention, (b) assaying a biological activity, and (b) determining if a biological activity of the polypeptide has been altered.

### **Targeted Delivery**

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In another embodiment, the invention provides a method of delivering compositions to targeted cells expressing a receptor for a polypeptide of the invention, or cells expressing a cell bound form of a polypeptide of the invention.

As discussed herein, polypeptides or antibodies of the invention may be associated with heterologous polypeptides, heterologous nucleic acids, toxins, or prodrugs via hydrophobic, hydrophilic, ionic and/or covalent interactions. In one embodiment, the invention provides a method for the specific delivery of compositions of the invention to cells by administering polypeptides of the invention (including antibodies) that are associated with heterologous polypeptides or nucleic acids. In one example, the invention provides a method for delivering a therapeutic protein into the targeted cell. In another example, the invention provides a method for delivering a single stranded nucleic acid (e.g., antisense or ribozymes) or double stranded nucleic acid (e.g., DNA that can integrate into the cell's genome or replicate episomally and that can be transcribed) into the targeted cell.

In another embodiment, the invention provides a method for the specific destruction of cells (e.g., the destruction of tumor cells) by administering polypeptides of the invention (e.g., polypeptides of the invention or antibodies of the invention) in association with toxins or cytotoxic prodrugs.

By "toxin" is meant compounds that bind and activate endogenous cytotoxic effector systems, radioisotopes, holotoxins, modified toxins, catalytic subunits of toxins, or any molecules or enzymes not normally present in or on the surface of a cell that under defined conditions cause the cell's death. Toxins that may be used according to the methods of the invention include, but are not limited to, radioisotopes known in the art, compounds such as, for example, antibodies (or complement fixing containing portions thereof) that bind an inherent or induced endogenous cytotoxic effector system, thymidine kinase, endonuclease, RNAse, alpha toxin, ricin, abrin, *Pseudomonas* exotoxin A, diphtheria toxin, saporin, momordin, gelonin, pokeweed antiviral protein, alpha-sarcin and cholera toxin. By "cytotoxic prodrug" is meant a non-toxic compound that is converted by an enzyme, normally present in the cell, into a cytotoxic compound. Cytotoxic prodrugs that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of benzoic acid mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxyacetamide derivatives of doxorubicin.

#### **Drug Screening**

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Further contemplated is the use of the polypeptides of the present invention, or the polypucleotides encoding these polypeptides, to screen for molecules which modify the activities of the polypeptides of the present invention. Such a method would include contacting the polypeptide of the present invention with a selected compound(s) suspected of having antagonist or agonist activity, and assaying the activity of these polypeptides following binding.

This invention is particularly useful for screening therapeutic compounds by using the polypeptides of the present invention, or binding fragments thereof, in any of a variety of drug screening techniques. The polypeptide or fragment employed in such a test may be affixed to a solid support, expressed on a cell surface, free in solution, or located intracellularly. One method of drug screening utilizes eukaryotic or prokaryotic host cells which are stably transformed with recombinant nucleic acids expressing the polypeptide or fragment. Drugs are screened against such transformed cells in competitive binding assays. One may measure, for example, the formulation of complexes between the agent being tested and a polypeptide of the present invention.

Thus, the present invention provides methods of screening for drugs or any other agents which affect activities mediated by the polypeptides of the present invention. These methods comprise contacting such an agent with a polypeptide of the present invention or a fragment thereof and assaying for the presence of a complex between the agent and the polypeptide or a fragment thereof, by methods well known in the art. In such a competitive binding assay, the agents to screen are typically labeled. Following incubation, free agent is separated from that present in bound form, and the amount of free or uncomplexed label is a measure of the ability of a particular agent to bind to the polypeptides of the present invention.

Another technique for drug screening provides high throughput screening for compounds having suitable binding affinity to the polypeptides of the present invention, and is described in great detail in European Patent Application 84/03564, published on September 13, 1984, which is incorporated herein by reference herein. Briefly stated, large numbers of different small peptide test compounds are synthesized on a solid substrate, such as plastic pins or some other surface. The peptide test compounds are reacted with polypeptides of the present invention and washed. Bound polypeptides are then detected by methods well known in the art. Purified polypeptides are coated directly onto plates for use in the aforementioned drug screening techniques. In addition, non-neutralizing antibodies may be used to capture the peptide and immobilize it on the solid support.

This invention also contemplates the use of competitive drug screening assays in which neutralizing antibodies capable of binding polypeptides of the present invention specifically compete with a test compound for binding to the polypeptides or fragments thereof. In this

manner, the antibodies are used to detect the presence of any peptide which shares one or more antigenic epitopes with a polypeptide of the invention.

## Antisense And Ribozyme (Antagonists)

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In specific embodiments, antagonists according to the present invention are nucleic acids corresponding to the sequences contained in SEQ ID NO:X, or the complementary strand thereof, and/or to cDNA sequences contained in cDNA ATCC Deposit No:Z identified for example, in Table 1A and/or 1B. In one embodiment, antisense sequence is generated internally, by the organism, in another embodiment, the antisense sequence is separately administered (see, for example, O'Connor, J., Neurochem. 56:560 (1991). Oligodeoxynucleotides as Antisense Inhibitors of Gene Expression, CRC Press, Boca Raton, FL (1988). Antisense technology can be used to control gene expression through antisense DNA or RNA, or through triple-helix formation. Antisense techniques are discussed for example, in Okano, J., Neurochem. 56:560 (1991); Oligodeoxynucleotides as Antisense Inhibitors of Gene Expression, CRC Press, Boca Raton, FL (1988). Triple helix formation is discussed in, for instance, Lee et al., Nucleic Acids Research 6:3073 (1979); Cooney et al., Science 241:456 (1988); and Dervan et al., Science 251:1300 (1991). The methods are based on binding of a polynucleotide to a complementary DNA or RNA.

For example, the use of c-myc and c-myb antisense RNA constructs to inhibit the growth of the non-lymphocytic leukemia cell line HL-60 and other cell lines was previously described. (Wickstrom et al. (1988); Anfossi et al. (1989)). These experiments were performed in vitro by incubating cells with the oligoribonucleotide. A similar procedure for *in vivo* use is described in WO 91/15580. Briefly, a pair of oligonucleotides for a given antisense RNA is produced as follows: A sequence complimentary to the first 15 bases of the open reading frame is flanked by an EcoR1 site on the 5 end and a HindIII site on the 3 end. Next, the pair of oligonucleotides is heated at 90°C for one minute and then annealed in 2X ligation buffer (20mM TRIS HCl pH 7.5, 10mM MgCl2, 10MM dithiothreitol (DTT) and 0.2 mM ATP) and then ligated to the EcoR1/Hind III site of the retroviral vector PMV7 (WO 91/15580).

For example, the 5' coding portion of a polynucleotide that encodes the polypeptide of the present invention may be used to design an antisense RNA oligonucleotide of from about 10 to 40 base pairs in length. A DNA oligonucleotide is designed to be complementary to a region of the gene involved in transcription thereby preventing transcription and the production of the receptor. The antisense RNA oligonucleotide hybridizes to the mRNA *in vivo* and blocks translation of the mRNA molecule into receptor polypeptide.

In one embodiment, the antisense nucleic acid of the invention is produced intracellularly by transcription from an exogenous sequence. For example, a vector or a portion thereof, is transcribed, producing an antisense nucleic acid (RNA) of the invention. Such a vector would contain a sequence encoding the antisense nucleic acid. Such a vector can remain episomal or

become chromosomally integrated, as long as it can be transcribed to produce the desired antisense RNA. Such vectors can be constructed by recombinant DNA technology methods standard in the art. Vectors can be plasmid, viral, or others known in the art, used for replication and expression in vertebrate cells. Expression of the sequence encoding the polypeptide of the present invention or fragments thereof, can be by any promoter known in the art to act in vertebrate, preferably human cells. Such promoters can be inducible or constitutive. Such promoters include, but are not limited to, the SV40 early promoter region (Bernoist and Chambon, Nature 29:304-310 (1981), the promoter contained in the 3' long terminal repeat of Rous sarcoma virus (Yamamoto et al., Cell 22:787-797 (1980), the herpes thymidine promoter (Wagner et al., Proc. Natl. Acad. Sci. U.S.A. 78:1441-1445 (1981), the regulatory sequences of the metallothionein gene (Brinster, et al., Nature 296:39-42 (1982)), etc.

The antisense nucleic acids of the invention comprise a sequence complementary to at least a portion of an RNA transcript of a gene of the present invention. However, absolute complementarity, although preferred, is not required. A sequence "complementary to at least a portion of an RNA," referred to herein, means a sequence having sufficient complementarity to be able to hybridize with the RNA, forming a stable duplex; in the case of double stranded antisense nucleic acids, a single strand of the duplex DNA may thus be tested, or triplex formation may be assayed. The ability to hybridize will depend on both the degree of complementarity and the length of the antisense nucleic acid. Generally, the larger the hybridizing nucleic acid, the more base mismatches with a RNA it may contain and still form a stable duplex (or triplex as the case may be). One skilled in the art can ascertain a tolerable degree of mismatch by use of standard procedures to determine the melting point of the hybridized complex.

Oligonucleotides that are complementary to the 5' end of the message, e.g., the 5' untranslated sequence up to and including the AUG initiation codon, should work most efficiently at inhibiting translation. However, sequences complementary to the 3' untranslated sequences of mRNAs have been shown to be effective at inhibiting translation of mRNAs as well. See generally, Wagner, R., 1994, Nature 372:333-335. Thus, oligonucleotides complementary to either the 5'- or 3'- non- translated, non-coding regions of polynucleotide sequences described herein could be used in an antisense approach to inhibit translation of endogenous mRNA. Oligonucleotides complementary to the 5' untranslated region of the mRNA should include the complement of the AUG start codon. Antisense oligonucleotides complementary to mRNA coding regions are less efficient inhibitors of translation but could be used in accordance with the invention. Whether designed to hybridize to the 5'-, 3'- or coding region of mRNA of the present invention, antisense nucleic acids should be at least six nucleotides in length. In specific aspects the oligonucleotide ranging from 6 to about 50 nucleotides, at least 25 nucleotides or at least 50 nucleotides.

The polynucleotides of the invention can be DNA or RNA or chimeric mixtures or derivatives or modified versions thereof, single-stranded or double-stranded. The oligonucleotide can be modified at the base moiety, sugar moiety, or phosphate backbone, for example, to improve stability of the molecule, hybridization, etc. The oligonucleotide may include other appended groups such as peptides (e.g., for targeting host cell receptors *in vivo*), or agents facilitating transport across the cell membrane (see, e.g., Letsinger et al., 1989, Proc. Natl. Acad. Sci. U.S.A. 86:6553-6556; Lemaitre et al., 1987, Proc. Natl. Acad. Sci. 84:648-652; PCT Publication No. WO88/09810, published December 15, 1988) or the blood-brain barrier (see, e.g., PCT Publication No. WO89/10134, published April 25, 1988), hybridization-triggered cleavage agents. (See, e.g., Krol et al., 1988, BioTechniques 6:958-976) or intercalating agents. (See, e.g., Zon, 1988, Pharm. Res. 5:539-549). To this end, the oligonucleotide may be conjugated to another molecule, e.g., a peptide, hybridization triggered cross-linking agent, transport agent, hybridization-triggered cleavage agent, etc.

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The antisense oligonucleotide may comprise at least one modified base moiety which is selected from the group including, but not limited to, 5-fluorouracil, 5-bromouracil, 5-chlorouracil, 5-iodouracil, hypoxanthine, xantine, 4-acetylcytosine, 5-(carboxyhydroxylmethyl) uracil, 5-carboxymethylaminomethyl-2-thiouridine, 5-carboxymethylaminomethyluracil, dihydrouracil, beta-D-galactosylqueosine, inosine, N6-isopentenyladenine, 1-methylguanine, 1-methylguanine, 2,2-dimethylguanine, 2-methyladenine, 2-methylguanine, 3-methylcytosine, 5-methylcytosine, N6-adenine, 7-methylguanine, 5-methylaminomethyluracil, 5-methoxyaminomethyl-2-thiouracil, beta-D-mannosylqueosine, 5'-methoxycarboxymethyluracil, 5-methoxyuracil, 2-methylthio-N6-isopentenyladenine, uracil-5-oxyacetic acid (v), wybutoxosine, pseudouracil, queosine, 2-thiocytosine, 5-methyl-2-thiouracil, 2-thiouracil, 4-thiouracil, 5-methyluracil, uracil-5-oxyacetic acid methylester, uracil-5-oxyacetic acid (v), 5-methyl-2-thiouracil, 3-(3-amino-3-N-2-carboxypropyl) uracil, (acp3)w, and 2,6-diaminopurine.

The antisense oligonucleotide may also comprise at least one modified sugar moiety selected from the group including, but not limited to, arabinose, 2-fluoroarabinose, xylulose, and hexose.

In yet another embodiment, the antisense oligonucleotide comprises at least one modified phosphate backbone selected from the group including, but not limited to, a phosphorothioate, a phosphorodithioate, a phosphoramidate, a phosphoramidate, a methylphosphonate, an alkyl phosphotriester, and a formacetal or analog thereof.

In yet another embodiment, the antisense oligonucleotide is an a-anomeric oligonucleotide. An a-anomeric oligonucleotide forms specific double-stranded hybrids with complementary RNA in which, contrary to the usual b-units, the strands run parallel to each other (Gautier et al., 1987, Nucl. Acids Res. 15:6625-6641). The oligonucleotide is a 2'-0-

methylribonucleotide (Inoue et al., 1987, Nucl. Acids Res. 15:6131-6148), or a chimeric RNA-DNA analogue (Inoue et al., 1987, FEBS Lett. 215:327-330).

Polynucleotides of the invention may be synthesized by standard methods known in the art, e.g. by use of an automated DNA synthesizer (such as are commercially available from Biosearch, Applied Biosystems, etc.). As examples, phosphorothioate oligonucleotides may be synthesized by the method of Stein et al. (1988, Nucl. Acids Res. 16:3209), methylphosphonate oligonucleotides can be prepared by use of controlled pore glass polymer supports (Sarin et al., 1988, Proc. Natl. Acad. Sci. U.S.A. 85:7448-7451), etc.

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While antisense nucleotides complementary to the coding region sequence could be used, those complementary to the transcribed untranslated region are most preferred.

Potential antagonists according to the invention also include catalytic RNA, or a ribozyme (See, e.g., PCT International Publication WO 90/11364, published October 4, 1990; Sarver et al, Science 247:1222-1225 (1990). While ribozymes that cleave mRNA at site specific recognition sequences can be used to destroy mRNAs, the use of hammerhead ribozymes is preferred. Hammerhead ribozymes cleave mRNAs at locations dictated by flanking regions that form complementary base pairs with the target mRNA. The sole requirement is that the target mRNA have the following sequence of two bases: 5'-UG-3'. The construction and production of hammerhead ribozymes is well known in the art and is described more fully in Haseloff and Gerlach, Nature 334:585-591 (1988). There are numerous potential hammerhead ribozyme cleavage sites within the nucleotide sequence of SEQ ID NO:X. Preferably, the ribozyme is engineered so that the cleavage recognition site is located near the 5' end of the mRNA; i.e., to increase efficiency and minimize the intracellular accumulation of non-functional mRNA transcripts.

As in the antisense approach, the ribozymes of the invention can be composed of modified oligonucleotides (e.g., for improved stability, targeting, etc.) and should be delivered to cells which express in vivo. DNA constructs encoding the ribozyme may be introduced into the cell in the same manner as described above for the introduction of antisense encoding DNA. A preferred method of delivery involves using a DNA construct "encoding" the ribozyme under the control of a strong constitutive promoter, such as, for example, pol III or pol II promoter, so that transfected cells will produce sufficient quantities of the ribozyme to destroy endogenous messages and inhibit translation. Since ribozymes unlike antisense molecules, are catalytic, a lower intracellular concentration is required for efficiency.

Antagonist/agonist compounds may be employed to inhibit the cell growth and proliferation effects of the polypeptides of the present invention on neoplastic cells and tissues, i.e. stimulation of angiogenesis of tumors, and, therefore, retard or prevent abnormal cellular growth and proliferation, for example, in tumor formation or growth.

The antagonist/agonist may also be employed to prevent hyper-vascular diseases, and prevent the proliferation of epithelial lens cells after extracapsular cataract surgery. Prevention of the mitogenic activity of the polypeptides of the present invention may also be desirous in cases such as restenosis after balloon angioplasty.

The antagonist/agonist may also be employed to prevent the growth of scar tissue during wound healing.

The antagonist/agonist may also be employed to treat the diseases described herein.

Thus, the invention provides a method of treating disorders or diseases, including but not limited to the disorders or diseases listed throughout this application, associated with overexpression of a polynucleotide of the present invention by administering to a patient (a) an antisense molecule directed to the polynucleotide of the present invention, and/or (b) a ribozyme directed to the polynucleotide of the present invention.

### **Binding Peptides and Other Molecules**

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The invention also encompasses screening methods for identifying polypeptides and nonpolypeptides that bind polypeptides of the invention, and the binding molecules identified thereby. These binding molecules are useful, for example, as agonists and antagonists of the polypeptides of the invention. Such agonists and antagonists can be used, in accordance with the invention, in the therapeutic embodiments described in detail, below.

This method comprises the steps of:

contacting polypeptides of the invention with a plurality of molecules; and identifying a molecule that binds the polypeptides of the invention.

The step of contacting the polypeptides of the invention with the plurality of molecules may be effected in a number of ways. For example, one may contemplate immobilizing the polypeptides on a solid support and bringing a solution of the plurality of molecules in contact with the immobilized polypeptides. Such a procedure would be akin to an affinity chromatographic process, with the affinity matrix being comprised of the immobilized polypeptides of the invention. The molecules having a selective affinity for the polypeptides can then be purified by affinity selection. The nature of the solid support, process for attachment of the polypeptides to the solid support, solvent, and conditions of the affinity isolation or selection are largely conventional and well known to those of ordinary skill in the art.

Alternatively, one may also separate a plurality of polypeptides into substantially separate fractions comprising a subset of or individual polypeptides. For instance, one can separate the plurality of polypeptides by gel electrophoresis, column chromatography, or like method known to those of ordinary skill for the separation of polypeptides. The individual polypeptides can also be produced by a transformed host cell in such a way as to be expressed on or about its outer surface (e.g., a recombinant phage). Individual isolates can then be "probed" by the polypeptides of the

invention, optionally in the presence of an inducer should one be required for expression, to determine if any selective affinity interaction takes place between the polypeptides and the individual clone. Prior to contacting the polypeptides with each fraction comprising individual polypeptides, the polypeptides could first be transferred to a solid support for additional convenience. Such a solid support may simply be a piece of filter membrane, such as one made of nitrocellulose or nylon. In this manner, positive clones could be identified from a collection of transformed host cells of an expression library, which harbor a DNA construct encoding a polypeptide having a selective affinity for polypeptides of the invention. Furthermore, the amino acid sequence of the polypeptide having a selective affinity for the polypeptides of the invention can be determined directly by conventional means or the coding sequence of the DNA encoding the polypeptide can frequently be determined more conveniently. The primary sequence can then be deduced from the corresponding DNA sequence. If the amino acid sequence is to be determined from the polypeptide itself, one may use microsequencing techniques. The sequencing technique may include mass spectroscopy.

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In certain situations, it may be desirable to wash away any unbound polypeptides from a mixture of the polypeptides of the invention and the plurality of polypeptides prior to attempting to determine or to detect the presence of a selective affinity interaction. Such a wash step may be particularly desirable when the polypeptides of the invention or the plurality of polypeptides are bound to a solid support.

The plurality of molecules provided according to this method may be provided by way of diversity libraries, such as random or combinatorial peptide or nonpeptide libraries which can be screened for molecules that specifically bind polypeptides of the invention. Many libraries are known in the art that can be used, e.g., chemically synthesized libraries, recombinant (e.g., phage display libraries), and in vitro translation-based libraries. Examples of chemically synthesized libraries are described in Fodor et al., 1991, Science 251:767-773; Houghten et al., 1991, Nature 354:84-86; Lam et al., 1991, Nature 354:82-84; Medynski, 1994, Bio/Technology 12:709-710;Gallop et al., 1994, J. Medicinal Chemistry 37(9):1233-1251; Ohlmeyer et al., 1993, Proc. Natl. Acad. Sci. USA 90:10922-10926; Erb et al., 1994, Proc. Natl. Acad. Sci. USA 91:11422-11426; Houghten et al., 1992, Biotechniques 13:412; Jayawickreme et al., 1994, Proc. Natl. Acad. Sci. USA 90:11708-11712; PCT Publication No. WO 93/20242; and Brenner and Lerner, 1992, Proc. Natl. Acad. Sci. USA 89:5381-5383.

Examples of phage display libraries are described in Scott and Smith, 1990, Science 249:386-390; Devlin et al., 1990, Science, 249:404-406; Christian, R. B., et al., 1992, J. Mol. Biol. 227:711-718); Lenstra, 1992, J. Immunol. Meth. 152:149-157; Kay et al., 1993, Gene 128:59-65; and PCT Publication No. WO 94/18318 dated Aug. 18, 1994.

In vitro translation-based libraries include but are not limited to those described in PCT

Publication No. WO 91/05058 dated Apr. 18, 1991; and Mattheakis et al., 1994, Proc. Natl. Acad. Sci. USA 91:9022-9026.

By way of examples of nonpeptide libraries, a benzodiazepine library (see e.g., Bunin et al., 1994, Proc. Natl. Acad. Sci. USA 91:4708-4712) can be adapted for use. Peptoid libraries (Simon et al., 1992, Proc. Natl. Acad. Sci. USA 89:9367-9371) can also be used. Another example of a library that can be used, in which the amide functionalities in peptides have been permethylated to generate a chemically transformed combinatorial library, is described by Ostresh et al. (1994, Proc. Natl. Acad. Sci. USA 91:11138-11142).

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The variety of non-peptide libraries that are useful in the present invention is great. For example, Ecker and Crooke, 1995, Bio/Technology 13:351-360 list benzodiazepines, hydantoins, piperazinediones, biphenyls, sugar analogs, beta-mercaptoketones, arylacetic acids, acylpiperidines, benzopyrans, cubanes, xanthines, aminimides, and oxazolones as among the chemical species that form the basis of various libraries.

Non-peptide libraries can be classified broadly into two types: decorated monomers and oligomers. Decorated monomer libraries employ a relatively simple scaffold structure upon which a variety functional groups is added. Often the scaffold will be a molecule with a known useful pharmacological activity. For example, the scaffold might be the benzodiazepine structure.

Non-peptide oligomer libraries utilize a large number of monomers that are assembled together in ways that create new shapes that depend on the order of the monomers. Among the monomer units that have been used are carbamates, pyrrolinones, and morpholinos. Peptoids, peptide-like oligomers in which the side chain is attached to the alpha amino group rather than the alpha carbon, form the basis of another version of non-peptide oligomer libraries. The first non-peptide oligomer libraries utilized a single type of monomer and thus contained a repeating backbone. Recent libraries have utilized more than one monomer, giving the libraries added flexibility.

Screening the libraries can be accomplished by any of a variety of commonly known methods. See, e.g., the following references, which disclose screening of peptide libraries: Parmley and Smith, 1989, Adv. Exp. Med. Biol. 251:215-218; Scott and Smith, 1990, Science 249:386-390; Fowlkes et al., 1992; BioTechniques 13:422-427; Oldenburg et al., 1992, Proc. Natl. Acad. Sci. USA 89:5393-5397; Yu et al., 1994, Cell 76:933-945; Staudt et al., 1988, Science 241:577-580; Bock et al., 1992, Nature 355:564-566; Tuerk et al., 1992, Proc. Natl. Acad. Sci. USA 89:6988-6992; Ellington et al., 1992, Nature 355:850-852; U.S. Pat. No. 5,096,815, U.S. Pat. No. 5,223,409, and U.S. Pat. No. 5,198,346, all to Ladner et al.; Rebar and Pabo, 1993, Science 263:671-673; and CT Publication No. WO 94/18318.

In a specific embodiment, screening to identify a molecule that binds polypeptides of the invention can be carried out by contacting the library members with polypeptides of the invention immobilized on a solid phase and harvesting those library members that bind to the polypeptides

of the invention. Examples of such screening methods, termed "panning" techniques are described by way of example in Parmley and Smith, 1988, Gene 73:305-318; Fowlkes et al., 1992, BioTechniques 13:422-427; PCT Publication No. WO 94/18318; and in references cited herein.

In another embodiment, the two-hybrid system for selecting interacting proteins in yeast (Fields and Song, 1989, Nature 340:245-246; Chien et al., 1991, Proc. Natl. Acad. Sci. USA 88:9578-9582) can be used to identify molecules that specifically bind to polypeptides of the invention.

Where the binding molecule is a polypeptide, the polypeptide can be conveniently selected from any peptide library, including random peptide libraries, combinatorial peptide libraries, or biased peptide libraries. The term "biased" is used herein to mean that the method of generating the library is manipulated so as to restrict one or more parameters that govern the diversity of the resulting collection of molecules, in this case peptides.

Thus, a truly random peptide library would generate a collection of peptides in which the probability of finding a particular amino acid at a given position of the peptide is the same for all 20 amino acids. A bias can be introduced into the library, however, by specifying, for example, that a lysine occur every fifth amino acid or that positions 4, 8, and 9 of a decapeptide library be fixed to include only arginine. Clearly, many types of biases can be contemplated, and the present invention is not restricted to any particular bias. Furthermore, the present invention contemplates specific types of peptide libraries, such as phage displayed peptide libraries and those that utilize a DNA construct comprising a lambda phage vector with a DNA insert.

As mentioned above, in the case of a binding molecule that is a polypeptide, the polypeptide may have about 6 to less than about 60 amino acid residues, preferably about 6 to about 10 amino acid residues, and most preferably, about 6 to about 22 amino acids. In another embodiment, a binding polypeptide has in the range of 15-100 amino acids, or 20-50 amino acids.

The selected binding polypeptide can be obtained by chemical synthesis or recombinant expression.

#### **Other Activities**

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A polypeptide, polynucleotide, agonist, or antagonist of the present invention, as a result of the ability to stimulate vascular endothelial cell growth, may be employed in treatment for stimulating re-vascularization of ischemic tissues due to various disease conditions such as thrombosis, arteriosclerosis, and other cardiovascular conditions. The polypeptide, polynucleotide, agonist, or antagonist of the present invention may also be employed to stimulate angiogenesis and limb regeneration, as discussed above.

A polypeptide, polynucleotide, agonist, or antagonist of the present invention may also be employed for treating wounds due to injuries, burns, post-operative tissue repair, and ulcers since

they are mitogenic to various cells of different origins, such as fibroblast cells and skeletal muscle cells, and therefore, facilitate the repair or replacement of damaged or diseased tissue.

A polypeptide, polynucleotide, agonist, or antagonist of the present invention may also be employed stimulate neuronal growth and to treat and prevent neuronal damage which occurs in certain neuronal disorders or neuro-degenerative conditions such as Alzheimer's disease, Parkinson's disease, and AIDS-related complex. A polypeptide, polynucleotide, agonist, or antagonist of the present invention may have the ability to stimulate chondrocyte growth, therefore, they may be employed to enhance bone and periodontal regeneration and aid in tissue transplants or bone grafts.

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A polypeptide, polynucleotide, agonist, or antagonist of the present invention may be also be employed to prevent skin aging due to sunburn by stimulating keratinocyte growth.

A polypeptide, polynucleotide, agonist, or antagonist of the present invention may also be employed for preventing hair loss, since FGF family members activate hair-forming cells and promotes melanocyte growth. Along the same lines, a polypeptide, polynucleotide, agonist, or antagonist of the present invention may be employed to stimulate growth and differentiation of hematopoietic cells and bone marrow cells when used in combination with other cytokines.

A polypeptide, polynucleotide, agonist, or antagonist of the present invention may also be employed to maintain organs before transplantation or for supporting cell culture of primary tissues. A polypeptide, polynucleotide, agonist, or antagonist of the present invention may also be employed for inducing tissue of mesodermal origin to differentiate in early embryos.

A polypeptide, polynucleotide, agonist, or antagonist of the present invention may also increase or decrease the differentiation or proliferation of embryonic stem cells, besides, as discussed above, hematopoietic lineage.

A polypeptide, polynucleotide, agonist, or antagonist of the present invention may also be used to modulate mammalian characteristics, such as body height, weight, hair color, eye color, skin, percentage of adipose tissue, pigmentation, size, and shape (e.g., cosmetic surgery). Similarly, a polypeptide, polynucleotide, agonist, or antagonist of the present invention may be used to modulate mammalian metabolism affecting catabolism, anabolism, processing, utilization, and storage of energy.

A polypeptide, polynucleotide, agonist, or antagonist of the present invention may be used to change a mammal's mental state or physical state by influencing biorhythms, caricadic rhythms, depression (including depressive disorders), tendency for violence, tolerance for pain, reproductive capabilities (preferably by Activin or Inhibin-like activity), hormonal or endocrine levels, appetite, libido, memory, stress, or other cognitive qualities.

A polypeptide, polynucleotide, agonist, or antagonist of the present invention may also be used as a food additive or preservative, such as to increase or decrease storage capabilities, fat content, lipid, protein, carbohydrate, vitamins, minerals, cofactors or other nutritional components.

The above-recited applications have uses in a wide variety of hosts. Such hosts include, but are not limited to, human, murine, rabbit, goat, guinea pig, camel, horse, mouse, rat, hamster, pig, micro-pig, chicken, goat, cow, sheep, dog, cat, non-human primate, and human. In specific embodiments, the host is a mouse, rabbit, goat, guinea pig, chicken, rat, hamster, pig, sheep, dog or cat. In preferred embodiments, the host is a mammal. In most preferred embodiments, the host is a human.

#### Other Preferred Embodiments

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Other preferred embodiments of the claimed invention include an isolated nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to a sequence of at least about 50 contiguous nucleotides in the nucleotide sequence of SEQ ID NO:X or the complementary strand thereto, the nucleotide sequence as defined in column 5 of Table 1B or columns 8 and 9 of Table 2 or the complementary strand thereto, and/or cDNA contained in ATCC Deposit No:Z.

Also preferred is a nucleic acid molecule wherein said sequence of contiguous nucleotides is included in the nucleotide sequence of the portion of SEQ ID NO:X as defined in column 5, "ORF (From-To)", in Table 1B.1.

Also preferred is a nucleic acid molecule wherein said sequence of contiguous nucleotides is included in the nucleotide sequence of the portion of SEQ ID NO:X as defined in columns 8 and 9, "NT From" and "NT To" respectively, in Table 2.

Also preferred is an isolated nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to a sequence of at least about 150 contiguous nucleotides in the nucleotide sequence of SEQ ID NO:X or the complementary strand thereto, the nucleotide sequence as defined in column 5 of Table 1B.1 or columns 8 and 9 of Table 2 or the complementary strand thereto, and/or cDNA contained in ATCC Deposit No:Z.

Further preferred is an isolated nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to a sequence of at least about 500 contiguous nucleotides in the nucleotide sequence of SEQ ID NO:X or the complementary strand thereto, the nucleotide sequence as defined in column 5 of Table 1B.1 or columns 8 and 9 of Table 2 or the complementary strand thereto, and/or cDNA contained in ATCC Deposit No:Z.

A further preferred embodiment is a nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to the nucleotide sequence of the portion of SEQ ID NO:X defined in column 5, "ORF (From-To)", in Table 1B.1.

A further preferred embodiment is a nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to the nucleotide sequence of the portion of SEQ ID NO:X defined in columns 8 and 9, "NT From" and "NT To", respectively, in Table 2.

A further preferred embodiment is an isolated nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to the complete nucleotide sequence of SEQ ID NO:X or the complementary strand thereto, the nucleotide sequence as defined in column 5 of Table 1B.1 or columns 8 and 9 of Table 2 or the complementary strand thereto, and/or cDNA contained in ATCC Deposit No:Z.

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Also preferred is an isolated nucleic acid molecule which hybridizes under stringent hybridization conditions to a nucleic acid molecule comprising a nucleotide sequence of SEQ ID NO:X or the complementary strand thereto, the nucleotide sequence as defined in column 5 of Table 1B.1 or columns 8 and 9 of Table 2 or the complementary strand thereto, and/or cDNA contained in ATCC Deposit No:Z, wherein said nucleic acid molecule which hybridizes does not hybridize under stringent hybridization conditions to a nucleic acid molecule having a nucleotide sequence consisting of only A residues or of only T residues.

Also preferred is a composition of matter comprising a DNA molecule which comprises the cDNA contained in ATCC Deposit No:Z.

Also preferred is an isolated nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to a sequence of at least 50 contiguous nucleotides of the cDNA sequence contained in ATCC Deposit No:Z.

Also preferred is an isolated nucleic acid molecule, wherein said sequence of at least 50 contiguous nucleotides is included in the nucleotide sequence of an open reading frame sequence encoded by cDNA contained in ATCC Deposit No:Z.

Also preferred is an isolated nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to sequence of at least 150 contiguous nucleotides in the nucleotide sequence encoded by cDNA contained in ATCC Deposit No:Z.

A further preferred embodiment is an isolated nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to sequence of at least 500 contiguous nucleotides in the nucleotide sequence encoded by cDNA contained in ATCC Deposit No:Z.

A further preferred embodiment is an isolated nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to the complete nucleotide sequence encoded by cDNA contained in ATCC Deposit No:Z.

A further preferred embodiment is a method for detecting in a biological sample a nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to a sequence of at least 50 contiguous nucleotides in a sequence selected from the group consisting of: a nucleotide sequence of SEQ ID NO:X or the complementary strand thereto; the nucleotide sequence as defined in column 5 of Table 1B.1 or columns 8 and 9 of Table 2 or the complementary strand thereto; and a nucleotide sequence encoded by cDNA contained in ATCC Deposit No:Z; which method comprises a step of comparing a nucleotide sequence of at least one nucleic acid molecule

in said sample with a sequence selected from said group and determining whether the sequence of said nucleic acid molecule in said sample is at least 95% identical to said selected sequence.

Also preferred is the above method wherein said step of comparing sequences comprises determining the extent of nucleic acid hybridization between nucleic acid molecules in said sample and a nucleic acid molecule comprising said sequence selected from said group. Similarly, also preferred is the above method wherein said step of comparing sequences is performed by comparing the nucleotide sequence determined from a nucleic acid molecule in said sample with said sequence selected from said group. The nucleic acid molecules can comprise DNA molecules or RNA molecules.

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A further preferred embodiment is a method for identifying the species, tissue or cell type of a biological sample which method comprises a step of detecting nucleic acid molecules in said sample, if any, comprising a nucleotide sequence that is at least 95% identical to a sequence of at least 50 contiguous nucleotides in a sequence selected from the group consisting of: a nucleotide sequence of SEQ ID NO:X or the complementary strand thereto; the nucleotide sequence as defined in column 5 of Table 1B.1 or columns 8 and 9 of Table 2 or the complementary strand thereto; and a nucleotide sequence of the cDNA contained in ATCC Deposit No:Z.

The method for identifying the species, tissue or cell type of a biological sample can comprise a step of detecting nucleic acid molecules comprising a nucleotide sequence in a panel of at least two nucleotide sequences, wherein at least one sequence in said panel is at least 95% identical to a sequence of at least 50 contiguous nucleotides in a sequence selected from said group.

Also preferred is a method for diagnosing in a subject a pathological condition associated with abnormal structure or expression of a nucleotide sequence of SEQ ID NO:X or the complementary strand thereto; the nucleotide sequence as defined in column 5 of Table 1B.1 or columns 8 and 9 of Table 2 or the complementary strand thereto; or the cDNA contained in ATCC Deposit No:Z which encodes a protein, wherein the method comprises a step of detecting in a biological sample obtained from said subject nucleic acid molecules, if any, comprising a nucleotide sequence that is at least 95% identical to a sequence of at least 50 contiguous nucleotides in a sequence selected from the group consisting of: a nucleotide sequence of SEQ ID NO:X or the complementary strand thereto; the nucleotide sequence as defined in column 5 of Table 1B.1 or columns 8 and 9 of Table 2 or the complementary strand thereto; and a nucleotide sequence of cDNA contained in ATCC Deposit No:Z.

The method for diagnosing a pathological condition can comprise a step of detecting nucleic acid molecules comprising a nucleotide sequence in a panel of at least two nucleotide sequences, wherein at least one sequence in said panel is at least 95% identical to a sequence of at least 50 contiguous nucleotides in a sequence selected from said group.

Also preferred is a composition of matter comprising isolated nucleic acid molecules wherein the nucleotide sequences of said nucleic acid molecules comprise a panel of at least two nucleotide sequences, wherein at least one sequence in said panel is at least 95% identical to a sequence of at least 50 contiguous nucleotides in a sequence selected from the group consisting of: a nucleotide sequence of SEQ ID NO:X or the complementary strand thereto; the nucleotide sequence as defined in column 5 of Table 1B.1 or columns 8 and 9 of Table 2 or the complementary strand thereto; and a nucleotide sequence encoded by cDNA contained in ATCC Deposit No:Z. The nucleic acid molecules can comprise DNA molecules or RNA molecules.

Also preferred is a composition of matter comprising isolated nucleic acid molecules wherein the nucleotide sequences of said nucleic acid molecules comprise a DNA microarray or "chip" of at least 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50, 100, 150, 200, 250, 300, 500, 1000, 2000, 3000, or 4000 nucleotide sequences, wherein at least one sequence in said DNA microarray or "chip" is at least 95% identical to a sequence of at least 50 contiguous nucleotides in a sequence selected from the group consisting of: a nucleotide sequence of SEQ ID NO:X wherein X is any integer as defined in Table 1A and/or 1B; and a nucleotide sequence encoded by a human cDNA clone identified by a cDNA "Clone ID" in Table 1A and/or 1B.

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Also preferred is an isolated polypeptide comprising an amino acid sequence at least 90% identical to a sequence of at least about 10 contiguous amino acids in the polypeptide sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and/or a polypeptide encoded by cDNA contained in ATCC Deposit No:Z.

Also preferred is an isolated polypeptide comprising an amino acid sequence at least 95% identical to a sequence of at least about 30 contiguous amino acids in the amino acid sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and/or a polypeptide encoded by cDNA contained in ATCC Deposit No:Z.

Further preferred is an isolated polypeptide comprising an amino acid sequence at least 95% identical to a sequence of at least about 100 contiguous amino acids in the amino acid sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and/or a polypeptide encoded by cDNA contained in ATCC Deposit No:Z.

Further preferred is an isolated polypeptide comprising an amino acid sequence at least 95% identical to the complete amino acid sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and/or a polypeptide encoded by cDNA contained in ATCC Deposit No:Z.

Further preferred is an isolated polypeptide comprising an amino acid sequence at least 90% identical to a sequence of at least about 10 contiguous amino acids in the complete amino acid sequence of a polypeptide encoded by contained in ATCC Deposit No:Z

Also preferred is a polypeptide wherein said sequence of contiguous amino acids is included in the amino acid sequence of a portion of said polypeptide encoded by cDNA contained in ATCC Deposit No:Z; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and/or the polypeptide sequence of SEQ ID NO:Y.

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Also preferred is an isolated polypeptide comprising an amino acid sequence at least 95% identical to a sequence of at least about 30 contiguous amino acids in the amino acid sequence of a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z.

Also preferred is an isolated polypeptide comprising an amino acid sequence at least 95% identical to a sequence of at least about 100 contiguous amino acids in the amino acid sequence of a polypeptide encoded by cDNA contained in ATCC Deposit No:Z.

Also preferred is an isolated polypeptide comprising an amino acid sequence at least 95% identical to the amino acid sequence of a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z.

Further preferred is an isolated antibody which binds specifically to a polypeptide comprising an amino acid sequence that is at least 90% identical to a sequence of at least 10 contiguous amino acids in a sequence selected from the group consisting of: a polypeptide sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z.

Further preferred is a method for detecting in a biological sample a polypeptide comprising an amino acid sequence which is at least 90% identical to a sequence of at least 10 contiguous amino acids in a sequence selected from the group consisting of: a polypeptide sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z; which method comprises a step of comparing an amino acid sequence of at least one polypeptide molecule in said sample with a sequence selected from said group and determining whether the sequence of said polypeptide molecule in said sample is at least 90% identical to said sequence of at least 10 contiguous amino acids.

Also preferred is the above method wherein said step of comparing an amino acid sequence of at least one polypeptide molecule in said sample with a sequence selected from said group comprises determining the extent of specific binding of polypeptides in said sample to an antibody which binds specifically to a polypeptide comprising an amino acid sequence that is at

least 90% identical to a sequence of at least 10 contiguous amino acids in a sequence selected from the group consisting of: a polypeptide sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z.

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Also preferred is the above method wherein said step of comparing sequences is performed by comparing the amino acid sequence determined from a polypeptide molecule in said sample with said sequence selected from said group.

Also preferred is a method for identifying the species, tissue or cell type of a biological sample which method comprises a step of detecting polypeptide molecules in said sample, if any, comprising an amino acid sequence that is at least 90% identical to a sequence of at least 10 contiguous amino acids in a sequence selected from the group consisting of: polypeptide sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z.

Also preferred is the above method for identifying the species, tissue or cell type of a biological sample, which method comprises a step of detecting polypeptide molecules comprising an amino acid sequence in a panel of at least two amino acid sequences, wherein at least one sequence in said panel is at least 90% identical to a sequence of at least 10 contiguous amino acids in a sequence selected from the above group.

Also preferred is a method for diagnosing in a subject a pathological condition associated with abnormal structure or expression of a nucleic acid sequence identified in Table 1A, 1B or Table 2 encoding a polypeptide, which method comprises a step of detecting in a biological sample obtained from said subject polypeptide molecules comprising an amino acid sequence in a panel of at least two amino acid sequences, wherein at least one sequence in said panel is at least 90% identical to a sequence of at least 10 contiguous amino acids in a sequence selected from the group consisting of: polypeptide sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z.

In any of these methods, the step of detecting said polypeptide molecules includes using an antibody.

Also preferred is an isolated nucleic acid molecule comprising a nucleotide sequence which is at least 95% identical to a nucleotide sequence encoding a polypeptide wherein said polypeptide comprises an amino acid sequence that is at least 90% identical to a sequence of at least 10 contiguous amino acids in a sequence selected from the group consisting of: polypeptide sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand

thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z.

Also preferred is an isolated nucleic acid molecule, wherein said nucleotide sequence encoding a polypeptide has been optimized for expression of said polypeptide in a prokaryotic host.

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Also preferred is a polypeptide molecule, wherein said polypeptide comprises an amino acid sequence selected from the group consisting of: polypeptide sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z.

Further preferred is a method of making a recombinant vector comprising inserting any of the above isolated nucleic acid molecule into a vector. Also preferred is the recombinant vector produced by this method. Also preferred is a method of making a recombinant host cell comprising introducing the vector into a host cell, as well as the recombinant host cell produced by this method.

Also preferred is a method of making an isolated polypeptide comprising culturing this recombinant host cell under conditions such that said polypeptide is expressed and recovering said polypeptide. Also preferred is this method of making an isolated polypeptide, wherein said recombinant host cell is a eukaryotic cell and said polypeptide is a human protein comprising an amino acid sequence selected from the group consisting of: polypeptide sequence of SEQ ID NO:Y; a polypeptide encoded by SEQ ID NO:X or the complementary strand thereto; the polypeptide encoded by the nucleotide sequence as defined in columns 8 and 9 of Table 2; and a polypeptide encoded by the cDNA contained in ATCC Deposit No:Z. The isolated polypeptide produced by this method is also preferred.

Also preferred is a method of treatment of an individual in need of an increased level of a protein activity, which method comprises administering to such an individual a Therapeutic comprising an amount of an isolated polypeptide, polynucleotide, immunogenic fragment or analogue thereof, binding agent, antibody, or antigen binding fragment of the claimed invention effective to increase the level of said protein activity in said individual.

Also preferred is a method of treatment of an individual in need of a decreased level of a protein activity, which method comprised administering to such an individual a Therapeutic comprising an amount of an isolated polypeptide, polynucleotide, immunogenic fragment or analogue thereof, binding agent, antibody, or antigen binding fragment of the claimed invention effective to decrease the level of said protein activity in said individual.

Also preferred is a method of treatment of an individual in need of a specific delivery of toxic compositions to diseased cells (e.g., tumors, leukemias or lymphomas), which method comprises administering to such an individual a Therapeutic comprising an amount of an isolated

polypeptide of the invention, including, but not limited to a binding agent, or antibody of the claimed invention that are associated with toxin or cytotoxic prodrugs.

Having generally described the invention, the same will be more readily understood by reference to the following examples, which are provided by way of illustration and are not intended as limiting.

## Description of Table 6

Table 6 summarizes some of the ATCC Deposits, Deposit dates, and ATCC designation numbers of deposits made with the ATCC in connection with the present application. These deposits were made in addition to those described in the Table 1A.

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TABLE		
ATCC Deposits	Deposit Date	ATCC Designation Number
LP01, LP02, LP03, LP04,	May-20-97	209059, 209060, 209061, 209062, 209063,
LP05, LP06, LP07, LP08,		209064, 209065, 209066, 209067, 209068,
LP09, LP10, LP11,		209069
LP12	Jan-12-98	209579
LP13	Jan-12-98	209578
LP14	Jul-16-98	203067
LP15	Jul-16-98	203068
LP16	Feb-1-99	203609
LP17	Feb-1-99	203610
LP20	Nov-17-98	203485
LP21	Jun-18-99	PTA-252
LP22	Jun-18-99	PTA-253
LP23	Dec-22-99	PTA-1081

15 Examples

#### Example 1: Isolation of a Selected cDNA Clone From the Deposited Sample

Each ATCC Deposit No:Z is contained in a plasmid vector. Table 7 identifies the vectors used to construct the cDNA library from which each clone was isolated. In many cases, the vector used to construct the library is a phage vector from which a plasmid has been excised. The following correlates the related plasmid for each phage vector used in constructing the cDNA library. For example, where a particular clone is identified in Table 7 as being isolated in the vector "Lambda Zap," the corresponding deposited clone is in "pBluescript."

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## Vector Used to Construct Library Corresponding Deposited Plasmid

Lambda Zap pBluescript (pBS)
Uni-Zap XR pBluescript (pBS)

Zap Express pBK

5 lafmid BA plafmid BA

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pSport1 pSport1

pCMVSport 2.0
pCMVSport 3.0
pCMVSport 3.0

pCR[®]2.1 pCR[®]2.1

Vectors Lambda Zap (U.S. Patent Nos. 5,128,256 and 5,286,636), Uni-Zap XR (U.S. Patent Nos. 5,128, 256 and 5,286,636), Zap Express (U.S. Patent Nos. 5,128,256 and 5,286,636), pBluescript (pBS) (Short, J. M. et al., Nucleic Acids Res. 16:7583-7600 (1988); Alting-Mees, M. A. and Short, J. M., Nucleic Acids Res. 17:9494 (1989)) and pBK (Alting-Mees, M. A. et al., Strategies 5:58-61 (1992)) are commercially available from Stratagene Cloning Systems, Inc., 11011 N. Torrey Pines Road, La Jolla, CA, 92037. pBS contains an ampicillin resistance gene and pBK contains a neomycin resistance gene. Both can be transformed into E. coli strain XL-1 Blue, also available from Stratagene. pBS comes in 4 forms SK+, SK-, KS+ and KS. The S and K refers to the orientation of the polylinker to the T7 and T3 primer sequences which flank the polylinker region ("S" is for SacI and "K" is for KpnI which are the first sites on each respective end of the linker). "+" or "-" refer to the orientation of the f1 origin of replication ("ori"), such that in one orientation, single stranded rescue initiated from the f1 ori generates sense strand DNA and in the other, antisense.

Vectors pSport1, pCMVSport 2.0 and pCMVSport 3.0, were obtained from Life Technologies, Inc., P. O. Box 6009, Gaithersburg, MD 20897. All Sport vectors contain an ampicillin resistance gene and may be transformed into E. coli strain DH10B, also available from Life Technologies. (See, for instance, Gruber, C. E., et al., Focus 15:59 (1993)). Vector lafmid BA (Bento Soares, Columbia University, NY) contains an ampicillin resistance gene and can be transformed into E. coli strain XL-1 Blue. Vector pCR®2.1, which is available from Invitrogen, 1600 Faraday Avenue, Carlsbad, CA 92008, contains an ampicillin resistance gene and may be transformed into E. coli strain DH10B, available from Life Technologies. (See, for instance, Clark, J. M., Nuc. Acids Res. 16:9677-9686 (1988) and Mead, D. et al., Bio/Technology 9: (1991)). Preferably, a polynucleotide of the present invention does not comprise the phage vector sequences identified for the particular clone in Table 7, as well as the corresponding plasmid vector sequences designated above.

The deposited material in the sample assigned the ATCC Deposit Number cited by reference to Table 1A, Table 2, Table 6 and Table 7 for any given cDNA clone also may contain one or more additional plasmids, each comprising a cDNA clone different from that given clone.

Thus, deposits sharing the same ATCC Deposit Number contain at least a plasmid for each ATCC Deposit No:Z.

# TABLE 7

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Libraries owned by Catalog	Catalog Description	Vector	ATCC Deposit
HUKA HUKB HUKC HUKD HUKE HUKF HUKG	Human Uterine Cancer	Lambda ZAP II	LP01
HCNA HCNB	Human Colon	Lambda Zap II	LP01
HFFA	Human Fetal Brain, random primed	Lambda Zap II	LP01
HTWA	Resting T-Cell	Lambda ZAP II	LP01
HBQA	Early Stage Human Brain, random primed	Lambda ZAP II	LP01
HLMB HLMF HLMG HLMH HLMI HLMJ HLMM HLMN	breast lymph node CDNA library	Lambda ZAP II	LP01
HCQA HCQB	human colon cancer	Lamda ZAP II	LP01
HMEA HMEC HMED HMEE HMEF HMEG HMEI HMEJ HMEK HMEL	Human Microvascular Endothelial Cells, fract. A	Lambda ZAP II	LP01
HUSA HUSC	Human Umbilical Vein Endothelial Cells, fract. A	Lambda ZAP II	LP01
HLQA HLQB	Hepatocellular Tumor	Lambda ZAP II	LP01
HHGA HHGB HHGC HHGD	Hemangiopericytoma	Lambda ZAP II	LP01
HSDM	Human Striatum Depression, re- rescue	Lambda ZAP II	LP01
HUSH	H Umbilical Vein Endothelial Cells, frac A, re-excision	Lambda ZAP II	LP01
HSGS	Salivary gland, subtracted	Lambda ZAP II	LP01
HFXA HFXB HFXC HFXD HFXE HFXF HFXG HFXH	Brain frontal cortex	Lambda ZAP II	LP01
HPQA HPQB HPQC	PERM TF274	Lambda ZAP II	LP01
HFXJ HFXK	Brain Frontal Cortex, re-excision		LP01
HCWA HCWB HCWC HCWD HCWE HCWF HCWG HCWH HCWI HCWJ HCWK	CD34 positive cells (Cord Blood)	ZAP Express	LP02
HCUA HCUB HCUC	CD34 depleted Buffy Coat (Cord Blood)	ZAP Express	LP02
HRSM	A-14 cell line	ZAP Express	LP02
HRSA	A1-CELL LINE	ZAP Express	LP02
HCUD HCUE HCUF HCUG HCUH HCUI	CD34 depleted Buffy Coat (Cord Blood), re-excision	ZAP Express	LP02
HBXE HBXF HBXG	H. Whole Brain #2, re-excision	ZAP Express	LP02
HRLM	L8 cell line	ZAP Express	LP02
НВХА НВХВ НВХС НВХО	Human Whole Brain #2 - Oligo dT > 1.5Kb	ZAP Express	LP02
HUDA HUDB HUDC	Testes	ZAP Express	LP02
ннтм ннто	H. hypothalamus, frac A;re- excision	ZAP Express	LP02
HHTL	H. hypothalamus, frac A	ZAP Express	LP02

Libraries owned by Catalog	Catalog Description	Vector	ATCC
Libraries owned by Catalog	Catalog Description	1 4 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6	Deposit
HASA HASD	Human Adult Spleen	Uni-ZAP XR	LP03
HFKC HFKD HFKE HFKF	Human Fetal Kidney	Uni-ZAP XR	LP03
HFKG	Trainer total retailoy		2.03
HE8A HE8B HE8C HE8D	Human 8 Week Whole Embryo	Uni-ZAP XR	LP03
HE8E HE8F HE8M HE8N			22.02
HGBA HGBD HGBE HGBF	Human Gall Bladder	Uni-ZAP XR	LP03
НСВС НСВН НСВІ	7		
HLHA HLHB HLHC HLHD	Human Fetal Lung III	Uni-ZAP XR	LP03
HILHE HILHF HILHG HILHH			
HLHQ	1		
HPMA HPMB HPMC HPMD	Human Placenta	Uni-ZAP XR	LP03
HPME HPMF HPMG HPMH	_		
HPRA HPRB HPRC HPRD	Human Prostate	Uni-ZAP XR	LP03
HSIA HSIC HSID HSIE	Human Adult Small Intestine	Uni-ZAP XR	LP03
HTEA HTEB HTEC HTED	Human Testes	Uni-ZAP XR	LP03
HTEE HTEF HTEG HTEH			
HTEI HTEJ HTEK			
НТРА HTPB HTPC HTPD	Human Pancreas Tumor	Uni-ZAP XR	LP03
HTPE.	<u> </u>		
HTTA HTTB HTTC HTTD	Human Testes Tumor	Uni-ZAP XR	LP03
HTTE HTTF			
НАРА НАРВ НАРС НАРМ	Human Adult Pulmonary	Uni-ZAP XR	LP03
HETA HETB HETC HETD	Human Endometrial Tumor	Uni-ZAP XR	LP03
HETE HETF HETG HETH			
HETI			
HHFB HHFC HHFD HHFE	Human Fetal Heart	Uni-ZAP XR	LP03
HHFF HHFG HHFH HHFI	<del></del>	** : 6 + 5 + 6	
HHPB HHPC HHPD HHPE	Human Hippocampus	Uni-ZAP XR	LP03
HHPF HHPG HHPH HCE1 HCE2 HCE3 HCE4	Human Cerebellum	Uni-ZAP XR	LP03
HCE5 HCEB HCEC HCED	Human Cerebellum	Uni-ZAP AR	LPU3
HCEE HCEF HCEG			
HUVB HUVC HUVD HUVE	Human Umbilical Vein, Endo.	Uni-ZAP XR	LP03
lievBhevehevBhevE	remake	OIII-ZAL ALK	E1 03
HSTA HSTB HSTC HSTD	Human Skin Tumor	Uni-ZAP XR	LP03
HTAA HTAB HTAC HTAD	Human Activated T-Cells	Uni-ZAP XR	LP03
HTAE			
HFEA HFEB HFEC	Human Fetal Epithelium (Skin)	Uni-ZAP XR	LP03
НЈРА НЈРВ НЈРС НЈР <b>D</b>	HUMAN JURKAT	Uni-ZAP XR	LP03
	MEMBRANE BOUND		ļ
i	POLYSOMES		į
HESA	Human epithelioid sarcoma	Uni-Zap XR	LP03
HLTA HLTB HLTC HLTD	Human T-Cell Lymphoma	Uni-ZAP XR	LP03
HLTE HLTF		·	
HFTA HFTB HFTC HFTD	Human Fetal Dura Mater	Uni-ZAP XR	LP03
HRDA HRDB HRDC HRDD	Human Rhabdomyosarcoma	Uni-ZAP XR	LP03
HRDE HRDF			j
HCAA HCAB HCAC	Cem cells cyclohexamide treated	Uni-ZAP XR	LP03
HRGA HRGB HRGC HRGD	Raji Cells, cyclohexamide	Uni-ZAP XR	LP03
	treated		
HSUA HSUB HSUC HSUM	Supt Cells, cyclohexamide	Uni-ZAP XR	LP03

Libraries owned by Catalog	Catalog Description	Vector	ATCC Deposit
	treated		
HT4A HT4C HT4D	Activated T-Cells, 12 hrs.	Uni-ZAP XR	LP03
HE9A HE9B HE9C HE9D HE9E HE9F HE9G HE9H HE9M HE9N	Nine Week Old Early Stage Human	Uni-ZAP XR	LP03
HATA HATB HATC HATD	Human Adrenal Gland Tumor	Uni-ZAP XR	LP03
TT5A	Activated T-Cells, 24 hrs.	Uni-ZAP XR	LP03
IFGA HFGM	Human Fetal Brain	Uni-ZAP XR	LP03
HNEA HNEB HNEC HNED	Human Neutrophil	Uni-ZAP XR	LP03
BGB HBGD	Human Primary Breast Cancer	Uni-ZAP XR	LP03
IBNA HBNB	Human Normal Breast	Uni-ZAP XR	LP03
HCAS	Cem Cells, cyclohexamide treated, subtra	Uni-ZAP XR	LP03
HHPS	Human Hippocampus, subtracted	pBS	LP03
HKCS HKCU	Human Colon Cancer, subtracted	pBS	LP03
HRGS	Raji cells, cyclohexamide treated, subtracted	pBS	LP03
HSUT	Supt cells, cyclohexamide treated, differentially expressed	pBS .	LP03
HT4S	Activated T-Cells, 12 hrs, subtracted	Uni-ZAP XR	LP03
HCDA HCDB HCDC HCDD HCDE	Human Chondrosarcoma	Uni-ZAP XR	LP03
HOAA HOAB HOAC	Human Osteosarcoma	Uni-ZAP XR	LP03
HTLA HTLB HTLC HTLD HTLE HTLF	Human adult testis, large inserts	Uni-ZAP XR	LP03
HLMA HLMC HLMD	Breast Lymph node cDNA library	Uni-ZAP XR	LP03
Н6ЕА Н6ЕВ Н6ЕС	HL-60, PMA 4H	Uni-ZAP XR	LP03
HTXA HTXB HTXC HTXD HTXE HTXF HTXG HTXH	Activated T-Cell (12hs)/Thiouridine labelledEco	Uni-ZAP XR	LP03
HNFA HNFB HNFC HNFD HNFE HNFF HNFG HNFH HNFJ	Human Neutrophil, Activated	Uni-ZAP XR	LP03
НТОВ НТОС	HUMAN TONSILS, FRACTION 2	Uni-ZAP XR	LP03
HMGB	Human OB MG63 control fraction I	Uni-ZAP XR	LP03
НОРВ	Human OB HOS control fraction	Uni-ZAP XR	LP03
HORB	Human OB HOS treated (10 nM E2) fraction I	Uni-ZAP XR	LP03
HSVA HSVB HSVC	Human Chronic Synovitis	Uni-ZAP XR	LP03
HROA	HUMAN STOMACH	Uni-ZAP XR	LP03
HBJA HBJB HBJC HBJD HBJE HBJF HBJG HBJH HBJI HBJJ	HUMAN B CELL LYMPHOMA	Uni-ZAP XR	LP03

Libraries owned by Catalog	Catalog Description	Vector	ATCC
Dioratios symbol by care-og			Deposit
нвік			
HCRA HCRB HCRC	human corpus colosum	Uni-ZAP XR	LP03
HODA HODB HODC HODD	human ovarian cancer	Uni-ZAP XR	LP03
HDSA	Dermatofibrosarcoma	Uni-ZAP XR	LP03
	Protuberance		2.05
HMWA HMWB HMWC	Bone Marrow Cell Line	Uni-ZAP XR	LP03
HMWD HMWE HMWF	(RS4;11)		
HMWG HMWH HMWI HMWJ			
HSOA	stomach cancer (human)	Uni-ZAP XR	LP03
HERA	SKIN	Uni-ZAP XR	LP03
HMDA	Brain-medulloblastoma	Uni-ZAP XR	LP03
HGLA HGLB HGLD	Glioblastoma	Uni-ZAP XR	LP03
HEAA	H. Atrophic Endometrium	Uni-ZAP XR	LP03
НВСА НВСВ	H. Lymph node breast Cancer	Uni-ZAP XR	LP03
HPWT	Human Prostate BPH, re-	Uni-ZAP XR	LP03
<b>                                     </b>	excision		~~~
HFVG HFVH HFVI	Fetal Liver, subtraction II	pBS	LP03
HNFI	Human Neutrophils, Activated,	pBS	LP03
	re-excision	P	
НВМВ НВМС НВМD	Human Bone Marrow, re-	pBS	LP03
	excision		
HKML HKMM HKMN	H. Kidney Medulla, re-excision	pBS	LP03
HKIX HKIY	H. Kidney Cortex, subtracted	pBS	LP03
HADT	H. Amygdala Depression,	pBS	LP03
	subtracted	•	
H6AS	Hl-60, untreated, subtracted	Uni-ZAP XR	LP03
H6ES	HL-60, PMA 4H, subtracted	Uni-ZAP XR	LP03
H6BS	HL-60, RA 4h, Subtracted	Uni-ZAP XR	LP03
H6CS	HL-60, PMA 1d, subtracted	Uni-ZAP XR	LP03
HTXJ HTXK	Activated T-	Uni-ZAP XR	LP03
	cell(12h)/Thiouridine-re-		
	excision		
HMSA HMSB HMSC HMSD	Monocyte activated	Uni-ZAP XR	LP03
HMSE HMSF HMSG HMSH			
HMSI HMSJ HMSK			
HAGA HAGB HAGC HAGD	Human Amygdala	Uni-ZAP XR	LP03
HAGE HAGF			7 700
HSRA HSRB HSRE	STROMAL -	Uni-ZAP XR	LP03
HODD HODE HODG HODI	OSTEOCLASTOMA	II : ZAD XO	T DO2
HSRD HSRF HSRG HSRH	Human Osteoclastoma Stromal	Uni-ZAP XR	LP03
HEOA HEOR HEOCHEOD	Cells - unamplified Stromal cell TF274	Uni-ZAP XR	LP03
HSQA HSQB HSQC HSQD	Stromai cell 1 F2/4	Uni-ZAP XK	LPUS
HSQE HSQF HSQG HSKA HSKB HSKC HSKD	Smooth muscle, serum treated	Uni-ZAP XR	LP03
HSKE HSKF HSKZ	Smooth muscle, setum treated	OIII-ZAF AK	15.03
HSLA HSLB HSLC HSLD	Smooth muscle,control	Uni-ZAP XR	LP03
HSLE HSLF HSLG	Jinoon muscie,condol	OIII-ZAF AK	11.05
HSDA HSDD HSDE HSDF	Spinal cord	Uni-ZAP XR	LP03
HSDG HSDH		Oni-Zau All	3
HPWS	Prostate-BPH subtracted II	pBS	LP03
M 410	1 TOSMIC-DI II SUULACICU II	1PDG	72. 43

Libraries owned by Catalog	Catalog Description	Vector	ATCC Deposit
HSKW HSKX HSKY	Smooth Muscle- HASTE normalized	pBS	LP03
HFPB HFPC HFPD	H. Frontal cortex,epileptic;re-	Uni-ZAP XR	LP03
HSDI HSDJ HSDK	Spinal Cord, re-excision	Uni-ZAP XR	LP03
HSKN HSKO		pBS	LP03
HSKG HSKH HSKI	Smooth muscle, serum induced,re-exc	pBS	LP03
HFCA HFCB HFCC HFCD HFCE HFCF	Human Fetal Brain	Uni-ZAP XR	LP04
HPTA HPTB HPTD	Human Pituitary	Uni-ZAP XR	LP04
НТНВ НТНС НТНD	Human Thymus	Uni-ZAP XR	LP04
HE6B HE6C HE6D HE6E HE6F HE6G HE6S	Human Whole Six Week Old Embryo	Uni-ZAP XR	LP04
HSSA HSSB HSSC HSSD HSSE HSSF HSSG HSSH HSSI HSSJ HSSK	Human Synovial Sarcoma	Uni-ZAP XR	LP04
НЕ7Т	7 Week Old Early Stage Human, subtracted	Uni-ZAP XR	LP04
НЕРА НЕРВ НЕРС	Human Epididymus	Uni-ZAP XR	LP04
HSNA HSNB HSNC HSNM HSNN	Human Synovium	Uni-ZAP XR	LP04
HPFB HPFC HPFD HPFE	Human Prostate Cancer, Stage C fraction	Uni-ZAP XR	LP04
HE2A HE2D HE2E HE2H HE2I HE2M HE2N HE2O	12 Week Old Early Stage Human	Uni-ZAP XR	LP04
HE2B HE2C HE2F HE2G HE2P HE2Q	12 Week Old Early Stage Human, II	Uni-ZAP XR	LP04
HPTS HPTT HPTU	Human Pituitary, subtracted	Uni-ZAP XR	LP04
HAUA HAUB HAUC	Amniotic Cells - TNF induced	Uni-ZAP XR	LP04
HAQA HAQB HAQC HAQD	Amniotic Cells - Primary Culture	Uni-ZAP XR	LP04
HWTA HWTB HWTC	wilm's tumor	Uni-ZAP XR	LP04
HBSD	Bone Cancer, re-excision	Uni-ZAP XR	LP04
HSGB	Salivary gland, re-excision	Uni-ZAP XR	LP04
HSJA HSJB HSJC	Smooth muscle-ILb induced	Uni-ZAP XR	LP04
HSXA HSXB HSXC HSXD	Human Substantia Nigra	Uni-ZAP XR	LP04
HSHA HSHB HSHC	Smooth muscle, IL1b induced	Uni-ZAP XR	LP04
HOUA HOUB HOUC HOUD HOUE	Adipocytes	Uni-ZAP XR	LP04
HPWA HPWB HPWC HPWD HPWE	Prostate BPH	Uni-ZAP XR	LP04
HELA HELB HELC HELD HELE HELF HELG HELH	Endothelial cells-control	Uni-ZAP XR	LP04
HEMA HEMB HEMC HEMD HEME HEMF HEMG HEMH	Endothelial-induced	Uni-ZAP XR	LP04
HBIA HBIB HBIC	Human Brain, Striatum	Uni-ZAP XR	LP04
HHSA HHSB HHSC HHSD HHSE	Human Hypothalmus, Schizophrenia	Uni-ZAP XR	LP04

Libraries owned by Catalog	Catalog Description	Vector	ATCC Deposit
HNGA HNGB HNGC HNGD HNGE HNGF HNGG HNGH HNGI HNGI	neutrophils control	Uni-ZAP XR	LP04
HNHA HNHB HNHC HNHD HNHE HNHF HNHG HNHH HNHI HNHI	Neutrophils IL-1 and LPS induced	Uni-ZAP XR	LP04
HSDB HSDC	STRIATUM DEPRESSION	Uni-ZAP XR	LP04
HHPT	Hypothalamus	Uni-ZAP XR	LP04
HSAT HSAU HSAV HSAW HSAX HSAY HSAZ	Anergic T-cell	Uni-ZAP XR	LP04
HBMS HBMT HBMU HBMV HBMW HBMX	Bone marrow	Uni-ZAP XR	LP04
HOEA HOEB HOEC HOED HOEE HOEF HOEJ	Osteoblasts	Uni-ZAP XR	LP04
HAIA HAIB HAIC HAID HAIE HAIF	Epithelial-TNFa and INF induced	Uni-ZAP XR	LP04
HTGA HTGB HTGC HTGD	Apoptotic T-cell	Uni-ZAP XR	LP04
HMCA HMCB HMCC HMCD HMCE	Macrophage-oxLDL	Uni-ZAP XR	LP04
HMAA HMAB HMAC HMAD HMAE HMAF HMAG	Macrophage (GM-CSF treated)	Uni-ZAP XR	LP04
НРНА	Normal Prostate	Uni-ZAP XR	LP04
HPIA HPIB HPIC	LNCAP prostate cell line	Uni-ZAP XR	LP04
НРЈА НРЈВ НРЈС	PC3 Prostate cell line	Uni-ZAP XR	LP04
HOSE HOSF HOSG	Human Osteoclastoma, re- excision	Uni-ZAP XR	LP04
HTGE HTGF	Apoptotic T-cell, re-excision	Uni-ZAP XR	LP04
HMAJ HMAK	H Macrophage (GM-CSF treated), re-excision	Uni-ZAP XR	LP04
HACB HACC HACD	Human Adipose Tissue, re- excision	Uni-ZAP XR	LP04
HFPA	H. Frontal Cortex, Epileptic	Uni-ZAP XR	LP04
HFAA HFAB HFAC HFAD HFAE	Alzheimer's, spongy change	Uni-ZAP XR	LP04
HFAM	Frontal Lobe, Dementia	Uni-ZAP XR	LP04
НМІА НМІВ НМІС	Human Manic Depression Tissue	Uni-ZAP XR	LP04
HTSA HTSE HTSF HTSG HTSH	Human Thymus	pBS	LP05
HPBA HPBB HPBC HPBD HPBE	Human Pineal Gland	pBS	LP05
HSAA HSAB HSAC	HSA 172 Cells	pBS	LP05
HSBA HSBB HSBC HSBM	HSC172 cells	pBS	LP05
<b>НЈАА НЈАВ НЈАС НЈА</b> Д	Jurkat T-cell G1 phase	pBS	LP05
ШВА НІВВ НІВС НІВD	Jurkat T-Cell, S phase	pBS	LP05
HAFA HAFB	Aorta endothelial cells + TNF-a	pBS	LP05
HAWA HAWB HAWC	Human White Adipose	pBS	LP05
HTNA HTNB	Human Thyroid	pBS	LP05
HONA	Normal Ovary, Premenopausal	pBS	LP05
HARA HARB	Human Adult Retina	pBS	LP05

Libraries owned by Catalog	Catalog Description	Vector	ATCC
			Deposit
HLJA HLJB	Human Lung	pCMVSport 1	LP06
HOFM HOFN HOFO	H. Ovarian Tumor, II, OV5232	pCMVSport 2.0	LP07
HOGA HOGB HOGC	OV 10-3-95	pCMVSport 2.0	LP07
HCGL	CD34+cells, II	pCMVSport 2.0	LP07
HDLA	Hodgkin's Lymphoma I	pCMVSport 2.0	LP07
HDTA HDTB HDTC HDTD	Hodgkin's Lymphoma II	pCMVSport 2.0	LP07
HDTE			
HKAA HKAB HKAC HKAD	Keratinocyte	pCMVSport2.0	LP07
HKAE HKAF HKAG HKAH			
HCIM	CAPFINDER, Crohn's Disease, lib 2	pCMVSport 2.0	LP07
HKAL	Keratinocyte, lib 2	pCMVSport2.0	LP07
HKAT	Keratinocyte, lib 3	pCMVSport2.0	LP07
HNDA	Nasal polyps	pCMVSport2.0	LP07
HDRA	H. Primary Dendritic Cells, lib 3	pCMVSport2.0	LP07
НОНА НОНВ НОНС	Human Osteoblasts II	pCMVSport2.0	LP07
HLDA HLDB HLDC	Liver, Hepatoma	pCMVSport3.0	LP08
HLDN HLDO HLDP	Human Liver, normal	pCMVSport3.0	LP08
НМТА	pBMC stimulated w/ poly I/C	pCMVSport3.0	LP08
HNTA	NTERA2, control	pCMVSport3.0	LP08
HDPA HDPB HDPC HDPD	Primary Dendritic Cells, lib 1	pCMVSport3.0	LP08
HDPF HDPG HDPH HDPI			
HDPJ HDPK			
HDPM HDPN HDPO HDPP	Primary Dendritic cells,frac 2	pCMVSport3.0	LP08
HMUA HMUB HMUC	Myoloid Progenitor Cell Line	pCMVSport3.0	LP08
HHEA HHEB HHEC HHED	T Cell helper I	pCMVSport3.0	LP08
HHEM HHEN HHEO HHEP	T cell helper II	pCMVSport3.0	LP08
HEQA HEQB HEQC	Human endometrial stromal cells	<u> </u>	LP08
НУМА НУМВ	Human endometrial stromal cells-treated with progesterone	pCMVSport3.0	LP08
HSWA HSWB HSWC	Human endometrial stromal cells-treated with estradiol	pCMVSport3.0	LP08
HSYA HSYB HSYC	Human Thymus Stromal Cells	pCMVSport3.0	LP08
HLWA HLWB HLWC	Human Placenta	pCMVSport3.0	LP08
HRAA HRAB HRAC	Rejected Kidney, lib 4	pCMVSport3.0	LP08
HMTM	PCR, pBMC I/C treated	PCRII	LP09
НМЈА	H. Meniingima, M6	pSport 1	LP10
HMKA HMKB HMKC HMKD HMKE	H. Meningima, M1	pSport 1	LP10
HUSG HUSI	Human umbilical vein endothelial cells, IL-4 induced	pSport 1	LP10
HUSX HUSY	Human Umbilical Vein Endothelial Cells, uninduced	pSport 1	LP10
HOFA	Ovarian Tumor I, OV5232	pSport 1	LP10
HCFA HCFB HCFC HCFD	T-Cell PHA 16 hrs	pSport 1	LP10
HCFL HCFM HCFN HCFO	T-Cell PHA 24 hrs	pSport 1	LP10
HADA HADC HADD HADE	Human Adipose	pSport 1	LP10
HADF HADG	•		
HOVA HOVB HOVC	Human Ovary	pSport 1	LP10

Libraries owned by Catalog	Catalog Description	Vector	ATCC
Ziorarios o vinos o y cararog	Samog Dosor.pao.		Deposit
HTWB HTWC HTWD HTWE HTWF	Resting T-Cell Library,II	pSport 1	LP10
НММА	Spleen metastic melanoma	pSport 1	LP10
HLYA HLYB HLYC HLYD HLYE	Spleen, Chronic lymphocytic leukemia	pSport 1	LP10
HCGA	CD34+ cell, I	pSport 1	LP10
HEOM HEON	Human Eosinophils	pSport 1	LP10
HTDA	Human Tonsil, Lib 3	pSport 1	LP10
HSPA	Salivary Gland, Lib 2	pSport 1	LP10
НСНА НСНВ НСНС	Breast Cancer cell line, MDA 36		LP10
НСНМ НСНО	Breast Cancer Cell line, angiogenic	pSport 1	LP10
HCIA	Crohn's Disease	pSport 1	LP10
HDAA HDAB HDAC	HEL cell line	pSport 1	LP10
НАВА	Human Astrocyte	pSport 1	LP10
HUFA HUFB HUFC	Ulcerative Colitis	pSport 1	LP10
HNTM	NTERA2 + retinoic acid, 14 days	pSport 1	LP10
HDQA	Primary Dendritic cells,CapFinder2, frac 1	pSport 1	LP10
HDQM	Primary Dendritic Cells, CapFinder, frac 2	pSport 1	LP10
HLDX	Human Liver, normal, CapFinder		LP10
HULA HULB HULC	Human Dermal Endothelial Cells, untreated	pSport1	LP10
HUMA	Human Dermal Endothelial cells,treated	pSport1	LP10
HCJA	Human Stromal Endometrial fibroblasts, untreated	pSport1	LP10
НСЈМ	Human Stromal endometrial fibroblasts, treated w/ estradiol	pSport1	LP10
HEDA	Human Stromal endometrial fibroblasts, treated with progesterone	pSport1	LP10
HFNA	Human ovary tumor cell OV350721	pSport1	LP10
HKGA HKGB HKGC HKGD	Merkel Cells	pSport1	LP10
HISA HISB HISC	Pancreas Islet Cell Tumor	pSport1	LP10
HLSA	Skin, burned	pSport1	LP10
HBZA	Prostate, BPH, Lib 2	pSport 1	LP10
HBZS	Prostate BPH,Lib 2, subtracted	pSport 1	LP10
HFIA HFIB HFIC	Synovial Fibroblasts (control)	pSport 1	LP10
HFIH HFII HFU	Synovial hypoxia	pSport 1	LP10
HFIT HFIU HFIV	Synovial IL-1/TNF stimulated	pSport 1	LP10
HGCA	Messangial cell, frac 1	pSport1	LP10
HMVA HMVB HMVC	Bone Marrow Stromal Cell, untreated	pSport1	LP10
HFIX HFIY HFIZ	Synovial Fibroblasts (II1/TNF), subt	pSport1	LP10
HFOX HFOY HFOZ	Synovial hypoxia-RSF	pSport1	LP10

Libraries owned by Catalog	Catalog Description	Vector	ATCC
Diolates of the same	James & James Paran		Deposit
	subtracted		1 1
HMQA HMQB HMQC HMQD	Human Activated Monocytes	Uni-ZAP XR	LP11
HLIA HLIB HLIC	Human Liver	pCMVSport 1	LP012
HHBA HHBB HHBC HHBD	Human Heart	pCMVSport 1	LP012
ННВЕ		Post	
НВВА НВВВ	Human Brain	pCMVSport 1	LP012
	Human Lung	pCMVSport 1	LP012
HOGA HOGB HOGC	Ovarian Tumor	pCMVSport 2.0	LP012
НТІМ	Human Tonsils, Lib 2	pCMVSport 2.0	LP012
HAMF HAMG	KMH2	pCMVSport 3.0	LP012
НАЈА НАЈВ НАЈС	L428	pCMVSport 3.0	LP012
HWBA HWBB HWBC HWBD	Dendritic cells, pooled	pCMVSport 3.0	LP012
HWBE	Donartic cons, pooled	por vopotro de	
HWAA HWAB HWAC HWAD	Human Bone Marrow, treated	pCMVSport 3.0	LP012
HWAE			
HYAA HYAB HYAC	B Cell lymphoma	pCMVSport 3.0	LP012
нwнс нwнн нwні	Healing groin wound, 6.5 hours post incision	pCMVSport 3.0	LP012
нwнр нwно нwнг	Healing groin wound; 7.5 hours post incision	pCMVSport 3.0	LP012
HARM	Healing groin wound - zero hr post-incision (control)	pCMVSport 3.0	LP012
HBIM	Olfactory epithelium; nasalcavity	pCMVSport 3.0	LP012
HWDA	Healing Abdomen wound; 70&90 min post incision	pCMVSport 3.0	LP012
HWEA	Healing Abdomen Wound;15 days post incision	pCMVSport 3.0	LP012
HWJA	Healing Abdomen Wound;21&29 days	pCMVSport 3.0	LP012
HNAL	Human Tongue, frac 2	pSport1	LP012
HMJA	H. Meniingima, M6	pSport1	LP012
HMKA HMKB HMKC HMKD	H. Meningima, M1	pSport1	LP012
HMKE			
HOFA	Ovarian Tumor I, OV5232	pSport1	LP012
HCFA HCFB HCFC HCFD	T-Cell PHA 16 hrs	pSport1	LP012
HCFL HCFM HCFN HCFO	T-Cell PHA 24 hrs	pSport1	LP012
НММА НММВ НММС	Spleen metastic melanoma	pSport1	LP012
HTDA	Human Tonsil, Lib 3	pSport1	LP012
HDBA	Human Fetal Thymus	pSport1	LP012
HDUA	Pericardium	pSport1	LP012
HBZA	Prostate, BPH, Lib 2	pSport1	LP012
HWCA	Larynx tumor	pSport1	LP012
HWKA	Normal lung	pSport1	LP012
HSMB	Bone marrow stroma,treated	pSport1	LP012
НВНМ	Normal trachea	pSport1	LP012
HLFC	Human Larynx	pSport1	LP012
HLRB	Siebben Polyposis	pSport1	LP012
HNIA	Mammary Gland	pSport1	LP012
HNJB	Palate carcinoma	pSport1	LP012

Catalog Description	Vector	ATCC
		Deposit
Palate normal	pSport1	LP012
Pharynx carcinoma	pSport1	LP012
Cheek Carcinoma	pSport1	LP012
Pharynx Carcinoma	pSport1	LP012
	pSport1	LP012
Pancreas normal PCA4 No		LP012
Tongue carcinoma		LP012
Human Uterine Cancer	Lambda ZAP II	LP013
Human Fetal Brain, random primed	Lambda ZAP II	LP013
Activated T-cell labeled with 4-thioluri	Lambda ZAP II	LP013
,,	Lambda ZAP II	LP013
Human microvascular	Lambda ZAP II	LP013
Human Umbilical Vein Endothelial cells, fract. A, re-	Lambda ZAP II	LP013
Hepatocellular tumor, re- excision	Lambda ZAP II	LP013
	Lambda ZAP II	LP013
Human Whole 6 week Old	pBluescript	LP013
Human Hippocampus, subtracted	pBluescript	LP013
LNCAP, differential expression	pBluescript	LP013
Early Stage Human Lung,	pBluescript	LP013
Supt cells, cyclohexamide	pBluescript	LP013
Supt cells, cyclohexamide	pBluescript	LP013
H. Striatum Depression, subtracted	pBluescript	LP013
Human Pituitary, Subtracted VII	pBluescript	LP013
H. Striatum Depression, subt II	pBluescript	LP013
H. Striatum Depression, subt	pBluescript	LP013
Human Pineal Gland	pBluescript SK-	LP013
Colorectal Tumor	pBluescript SK-	LP013
	pBluescript SK-	LP013
	pBluescript SK-	LP013
Jurkat T-cell, S1 phase	pBluescript SK-	LP013
		LP013
		LP013
		LP013
Human Fetal Brain	Uni-ZAP XR	LP013
	1	1
	Palate normal Pharynx carcinoma Cheek Carcinoma Pharynx Carcinoma Larynx Carcinoma Pancreas normal PCA4 No Tongue carcinoma Human Uterine Cancer  Human Fetal Brain, random primed Activated T-cell labeled with 4- thioluri Early Stage Human Brain, random primed Human microvascular Endothelial cells, fract. B Human Umbilical Vein Endothelial cells, fract. A, re- excision Hepatocellular tumor, re- excision Resting T-cell, re-excision Human Whole 6 week Old Embryo (II), subt Human Hippocampus, subtracted LNCAP, differential expression Early Stage Human Lung, Subtracted Supt cells, cyclohexamide treated, subtracted Supt cells, cyclohexamide treated, differentially expressed H. Striatum Depression, subtracted Human Pituitary, Subtracted VII H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt Human Pineal Gland Colorectal Tumor HSC172 cells Jurkat T-cell, S1 phase Human Thyroid Human Adult Heart Whole 6 week Old Embryo	Palate normal pSport1 Pharynx carcinoma pSport1 Pharynx Carcinoma pSport1 Pharynx Carcinoma pSport1 Larynx Carcinoma pSport1 Pancreas normal PCA4 No pSport1 Pancreas normal PCA4 No pSport1 Tongue carcinoma pSport1 Human Uterine Cancer Lambda ZAP II Human Fetal Brain, random primed Activated T-cell labeled with 4-thioluri Early Stage Human Brain, random primed Human microvascular Lambda ZAP II Endothelial cells, fract. B Human Umbilical Vein Endothelial cells, fract. A, re-excision Hepatocellular tumor, re-excision Resting T-cell, re-excision Lambda ZAP II Human Whole 6 week Old Embryo (II), subt Human Hippocampus, pBluescript Early Stage Human Lung, pBluescript Subtracted LNCAP, differential expression pBluescript Early Stage Human Lung, pBluescript Early Stage Human Lung, pBluescript Treated, subtracted Supt cells, cyclohexamide treated, subtracted H. Striatum Depression, pBluescript Human Pinuitary, Subtracted VII H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt II H. Striatum Depression, subt

Libraries owned by Catalog	Catalog Description	Vector	ATCC Deposit
HFKG	·	•	
HGBA HGBD HGBE HGBF	Human Gall Bladder	Uni-ZAP XR	LP013
HGBG	·		
	Human Prostate	Uni-ZAP XR	LP013
	Human Testes	Uni-ZAP XR	LP013
НТЕЕ			
·	Human Testes Tumor	Uni-ZAP XR	LP013
HTTE			-
НҮВА НҮВВ	Human Fetal Bone	Uni-ZAP XR	LP013
HFLA	Human Fetal Liver	Uni-ZAP XR	LP013
HHFB HHFC HHFD HHFE	Human Fetal Heart	Uni-ZAP XR	LP013
HHFF			
HUVB HUVC HUVD HUVE	Human Umbilical Vein, End. remake	Uni-ZAP XR	LP013
НТНВ НТНС НТНD	Human Thymus	Uni-ZAP XR	LP013
HSTA HSTB HSTC HSTD	Human Skin Tumor	Uni-ZAP XR	LP013
HTAA HTAB HTAC HTAD	Human Activated T-cells	Uni-ZAP XR	LP013
НТАЕ			
HFEA HFEB HFEC	Human Fetal Epithelium (skin)	Uni-ZAP XR	LP013
НЈРА НЈРВ НЈРС НЈРО	Human Jurkat Membrane Bound	Uni-ZAP XR	LP013
	Polysomes		
HESA	Human Epithelioid Sarcoma	Uni-ZAP XR	LP013
HALS	Human Adult Liver, Subtracted	Uni-ZAP XR	LP013
HFTA HFTB HFTC HFTD	Human Fetal Dura Mater	Uni-ZAP XR	LP013
НСАА НСАВ НСАС	Cem cells, cyclohexamide treated	Uni-ZAP XR	LP013
HRGA HRGB HRGC HRGD	Raji Cells, cyclohexamide treated	Uni-ZAP XR	LP013
НЕ9А НЕ9В НЕ9С НЕ9D	Nine Week Old Early Stage	Uni-ZAP XR	LP013
HE9E	Human		1
HSFA	Human Fibrosarcoma	Uni-ZAP XR	LP013
HATA HATB HATC HATD	Human Adrenal Gland Tumor	Uni-ZAP XR	LP013
HATE			
HTRA	Human Trachea Tumor	Uni-ZAP XR	LP013
HE2A HE2D HE2E HE2H HE2I	12 Week Old Early Stage Human	Uni-ZAP XR	LP013
HE2B HE2C HE2F HE2G HE2P	12 Week Old Early Stage Human, II	Uni-ZAP XR	LP013
HNEA HNEB HNEC HNED HNEE	Human Neutrophil	Uni-ZAP XR	LP013
HBGA	Human Primary Breast Cancer	Uni-ZAP XR	LP013
HPTS HPTT HPTU	Human Pituitary, subtracted	Uni-ZAP XR	LP013
HMQA HMQB HMQC HMQD	Human Activated Monocytes	Uni-ZAP XR	LP013
HOAA HOAB HOAC	Human Osteosarcoma	Uni-ZAP XR	LP013
HTOA HTOD HTOE HTOF	human tonsils	Uni-ZAP XR	LP013
HTOG	The state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the state of the s		
HMGB	Human OB MG63 control fraction I	Uni-ZAP XR	LP013
НОРВ	Human OB HOS control fraction	Uni-ZAP XR	LP013
	lt .		

Libraries owned by Catalog	Catalog Description	Vector	ATCC
	TO 6 T		Deposit
	E2) fraction I	II. ZAD VD	7 7010
HAUA HAUB HAUC	Amniotic Cells - TNF induced	Uni-ZAP XR	LP013
HAQA HAQB HAQC HAQD	Amniotic Cells - Primary Culture	Uni-ZAP XR	LP013
HROA HROC	HUMAN STOMACH	Uni-ZAP XR	LP013
НВЈА НВЈВ НВЈС НВЈО НВЈЕ	HUMAN B CELL LYMPHOMA	Uni-ZAP XR	LP013
HODA HODB HODC HODD	human ovarian cancer	Uni-ZAP XR	LP013
HCPA	Corpus Callosum	Uni-ZAP XR	LP013
HSOA	stomach cancer (human)	Uni-ZAP XR	LP013
HERA	SKIN	Uni-ZAP XR	LP013
HMDA	Brain-medulloblastoma	Uni-ZAP XR	LP013
HGLA HGLB HGLD	Glioblastoma	Uni-ZAP XR	LP013
HWTA HWTB HWTC	wilm's tumor	Uni-ZAP XR	LP013
HEAA	H. Atrophic Endometrium	Uni-ZAP XR	LP013
HAPN HAPO HAPP HAPQ HAPR	Human Adult Pulmonary;re-	Uni-ZAP XR	LP013
HLTG HLTH	Human T-cell lymphoma;re- excision	Uni-ZAP XR	LP013
HAHC HAHD HAHE	Human Adult Heart;re-excision	Uni-ZAP XR	LP013
HAGA HAGB HAGC HAGD HAGE	Human Amygdala	Uni-ZAP XR	LP013
HSJA HSJB HSJC	Smooth muscle-ILb induced	Uni-ZAP XR	LP013
HSHA HSHB HSHC	Smooth muscle, IL1b induced	Uni-ZAP XR	LP013
HPWA HPWB HPWC HPWD HPWE	Prostate BPH	Uni-ZAP XR	LP013
HPIA HPIB HPIC	LNCAP prostate cell line	Uni-ZAP XR	LP013
НРЈА НРЈВ НРЈС	PC3 Prostate cell line	Uni-ZAP XR	LP013
НВТА	Bone Marrow Stroma, TNF&LPS ind	Uni-ZAP XR	LP013
HMCF HMCG HMCH HMCI HMCJ	Macrophage-oxLDL; re-excision	Uni-ZAP XR	LP013
HAGG HAGH HAGI	Human Amygdala;re-excision	Uni-ZAP XR	LP013
HACA	H. Adipose Tissue	Uni-ZAP XR	LP013
HKFB	K562 + PMA (36 hrs),re- excision	ZAP Express	LP013
HCWT HCWU HCWV	CD34 positive cells (cord blood),re-ex	ZAP Express	LP013
HBWA	Whole brain	ZAP Express	LP013
НВХА НВХВ НВХС НВХО	Human Whole Brain #2 - Oligo dT > 1.5Kb	ZAP Express	LP013
HAVM	Temporal cortex-Alzheizmer	pT-Adv	LP014
HAVT	Hippocampus, Alzheimer Subtracted	pT-Adv	LP014
HHAS	CHME Cell Line	Uni-ZAP XR	LP014
HAJR	Larynx normal	pSport 1	LP014
HWLE HWLF HWLG HWLH	Colon Normal	pSport 1	LP014
HCRM HCRN HCRO	Colon Carcinoma	pSport 1	LP014
HWLI HWLJ HWLK	Colon Normal	pSport 1	LP014
HWLQ HWLR HWLS HWLT	Colon Tumor	pSport 1	LP014

Libraries owned by Catalog	Catalog Description	Vector	ATCC
Libraries owned by Catalog	Catalog Doscription	Voctor	Deposit
HBFM	Gastrocnemius Muscle	pSport 1	LP014
HBOD HBOE	Quadriceps Muscle	pSport 1	LP014
HBKD HBKE	Soleus Muscle	pSport 1	LP014
HCCM	Pancreatic Langerhans	pSport 1	LP014
HWGA	Larynx carcinoma	pSport 1	LP014
HWGM HWGN	Larynx carcinoma	pSport 1	LP014
HWLA HWLB HWLC	Normal colon	pSport 1	LP014 LP014
	Colon Tumor	pSport 1	LP014
HWLM HWLN	Pancreas Tumor	pSport 1	LP014 LP014
HVAM HVAN HVAO			LP014 LP014
HWGQ	Larynx carcinoma	pSport 1	LP014 LP014
HAQM HAQN	Salivary Gland	pSport 1	
HASM	Stomach; normal	pSport 1	LP014
HBCM	Uterus; normal	pSport 1	LP014
HCDM	Testis; normal	pSport 1	LP014
HDJM	Brain; normal	pSport 1	LP014
HEFM	Adrenal Gland, normal	pSport 1	LP014
HBAA	Rectum normal	pSport 1	LP014
HFDM	Rectum tumour	pSport 1	LP014
HGAM	Colon, normal	pSport 1	LP014
HHMM	Colon, tumour	pSport 1	LP014
HCLB HCLC	Human Lung Cancer	Lambda Zap II	LP015
HRLA	L1 Cell line	ZAP Express	LP015
ННАМ	Hypothalamus, Alzheimer's	pCMVSport 3.0	LP015
HKBA	Ku 812F Basophils Line	pSport 1	LP015
HS2S	Saos2, Dexamethosome Treated	pSport 1	LP016
HA5A	Lung Carcinoma A549	pSport 1	LP016
	TNFalpha activated		7 7016
HTFM	TF-1 Cell Line GM-CSF Treated		LP016
HYAS	Thyroid Tumour	pSport 1	LP016
HUTS	Larynx Normal	pSport 1	LP016
HXOA	Larynx Tumor	pSport 1	LP016
HEAH	Ea.hy.926 cell line	pSport 1	LP016
HINA	Adenocarcinoma Human	pSport 1	LP016
HRMA	Lung Mesothelium	pSport 1	LP016
HLCL	Human Pre-Differentiated Adipocytes	Uni-Zap XR	LP017
HS2A	Saos2 Cells	pSport 1	LP020
HS2I	Saos2 Cells; Vitamin D3 Treated	pSport 1	LP020
HUCM	CHME Cell Line, untreated	pSport 1	LP020
HEPN	Aryepiglottis Normal	pSport 1	LP020
HPSN	Sinus Piniformis Tumour	pSport 1	LP020
HNSA	Stomach Normal	pSport 1	LP020
HNSM	Stomach Tumour	pSport 1	LP020
HNLA	Liver Normal Met5No	pSport 1	LP020
HUTA	Liver Tumour Met 5 Tu	pSport 1	LP020
HOCN	Colon Normal	pSport 1	LP020
HOCT	Colon Tumor	pSport 1	LP020
HTNT	Tongue Tumour	pSport 1	LP020
HLXN	Larynx Normal	pSport 1	LP020
HLXT	Larynx Tumour	pSport 1	LP020
IILAI	Irai Any I million	lhohoir i	121 020

Libraries owned by Catalog	Catalog Description	Vector	ATCC
			Deposit
HTYN	Thymus	pSport 1	LP020
HPLN	Placenta	pSport 1	LP020
HTNG	Tongue Normal	pSport 1	LP020
HZAA	Thyroid Normal (SDCA2 No)	pSport 1	LP020
HWES	Thyroid Thyroiditis	pSport 1	LP020
HFHD	Ficolled Human Stromal Cells,	pTrip1Ex2	LP021
	5Fu treated	F	•
HFHM,HFHN	Ficolled Human Stromal Cells,	pTrip1Ex2	LP021
	Untreated	P	
HPCI	Hep G2 Cells, lambda library	lambda Zap-CMV XR	LP021
НВСА,НВСВ,НВСС	H. Lymph node breast Cancer	Uni-ZAP XR	LP021
НСОК	Chondrocytes	pSPORT1	LP022
HDCA, HDCB, HDCC	Dendritic Cells From CD34	pSPORT1	LP022
TIDEN, TIDED, TIDEE	Cells	POLOXILL	0
HDMA, HDMB	CD40 activated monocyte	pSPORT1	LP022
	dendritic cells		
HDDM, HDDN, HDDO	LPS activated derived dendritic	pSPORT1	LP022
	cells	1	
HPCR	Hep G2 Cells, PCR library	lambda Zap-CMV	LP022
	•	XR	
HAAA, HAAB, HAAC	Lung, Cancer (4005313A3):	pSPORT1	LP022
	Invasive Poorly Differentiated	- 	
	Lung Adenocarcinoma		
HIPA, HIPB, HIPC	Lung, Cancer (4005163 B7):	pSPORT1	LP022
	Invasive, Poorly Diff.		
	Adenocarcinoma, Metastatic		i
HOOH, HOOI	Ovary, Cancer: (4004562 B6)	pSPORT1	LP022
	Papillary Serous Cystic		
	Neoplasm, Low Malignant Pot		
HIDA	Lung, Normal: (4005313 B1)	pSPORT1	LP022
HUJA,HUJB,HUJC,HUJD,HUJ	B-Cells	pCMVSport 3.0	LP022
E			
HNOA,HNOB,HNOC,HNOD	Ovary, Normal: (9805C040R)	pSPORT1	LP022
HNLM	Lung, Normal: (4005313 B1)	pSPORT1	LP022
HSCL	Stromal Cells	pSPORT1	LP022
HAAX	Lung, Cancer: (4005313 A3)	pSPORT1	LP022
	Invasive Poorly-differentiated	Ī	
	Metastatic lung adenocarcinoma		
HUUA,HUUB,HUUC,HUUD	B-cells (unstimulated)	pTrip1Ex2	LP022
HWWA,HWWB,HWWC,HWW	B-cells (stimulated)	pSPORT1	LP022
D,HWWE,HWWF,HWWG			<u> </u>
HCCC	Colon, Cancer: (9808C064R)	pCMVSport 3.0	LP023
HPDO HPDP HPDQ HPDR	Ovary, Cancer (9809C332):	pSport 1	LP023
HPD	Poorly differentiated	ļ ⁻	
	adenocarcinoma	_	
HPCO HPCP HPCQ HPCT	Ovary, Cancer (15395A1F):	pSport 1	LP023
	Grade II Papillary Carcinoma		
HOCM HOCO HOCP HOCQ	Ovary, Cancer: (15799A1F)	pSport 1	LP023
	Poorly differentiated carcinoma	, ,	
HCBM HCBN HCBO	Breast, Cancer: (4004943 A5)	pSport 1	LP023
		4	<u> </u>

Catalog Description	Vector	ATCC
		Deposit
Breast, Normal: (4005522B2)	pSport 1	LP023
Breast, Cancer: (4005522 A2)	pSport 1	LP023
Breast, Cancer: (9806C012R)	pSport 1	LP023
Stromal cells 3.88	pSport 1	LP023
Ovary, Cancer: (4004332 A2)	pSport 1	LP023
Stromal cells (HBM3.18)	pSport 1	LP023
stromal cell clone 2.5	pSport 1	LP023
Breast Cancer: (4005385 A2)	pSport 1	LP023
Ovary, Cancer (4004650 A3):	pSport 1	LP023
Well-Differentiated		
Micropapillary Serous	1	1
Carcinoma	<u> </u>	
Breast, Cancer: (9802C020E)	pSport 1	LP023
Human Bone Marrow, treated	pSport 1	LP023
	Breast, Normal: (4005522B2) Breast, Cancer: (4005522 A2) Breast, Cancer: (9806C012R) Stromal cells 3.88 Ovary, Cancer: (4004332 A2) Stromal cells (HBM3.18) stromal cell clone 2.5 Breast Cancer: (4005385 A2) Ovary, Cancer (4004650 A3): Well-Differentiated Micropapillary Serous Carcinoma Breast, Cancer: (9802C020E)	Breast, Normal: (4005522B2) pSport 1 Breast, Cancer: (4005522 A2) pSport 1 Breast, Cancer: (9806C012R) pSport 1 Stromal cells 3.88 pSport 1 Ovary, Cancer: (4004332 A2) pSport 1 Stromal cells (HBM3.18) pSport 1 stromal cell clone 2.5 pSport 1 Breast Cancer: (4005385 A2) pSport 1 Ovary, Cancer (4004650 A3): pSport 1 Well-Differentiated Micropapillary Serous Carcinoma Breast, Cancer: (9802C020E) pSport 1

Two nonlimiting examples are provided below for isolating a particular clone from the deposited sample of plasmid cDNAs cited for that clone in Table 7. First, a plasmid is directly isolated by screening the clones using a polynucleotide probe corresponding to the nucleotide sequence of SEQ ID NO:X.

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Particularly, a specific polynucleotide with 30-40 nucleotides is synthesized using an Applied Biosystems DNA synthesizer according to the sequence reported. The oligonucleotide is labeled, for instance, with ³²P-γ-ATP using T4 polynucleotide kinase and purified according to routine methods. (E.g., Maniatis et al., Molecular Cloning: A Laboratory Manual, Cold Spring Harbor Press, Cold Spring, NY (1982)). The plasmid mixture is transformed into a suitable host, as indicated above (such as XL-1 Blue (Stratagene)) using techniques known to those of skill in the art, such as those provided by the vector supplier or in related publications or patents cited above. The transformants are plated on 1.5% agar plates (containing the appropriate selection agent, e.g., ampicillin) to a density of about 150 transformants (colonies) per plate. These plates are screened using Nylon membranes according to routine methods for bacterial colony screening (e.g., Sambrook et al., Molecular Cloning: A Laboratory Manual, 2nd Edit., (1989), Cold Spring Harbor Laboratory Press, pages 1.93 to 1.104), or other techniques known to those of skill in the art.

Alternatively, two primers of 17-20 nucleotides derived from both ends of the nucleotide sequence of SEQ ID NO:X are synthesized and used to amplify the desired cDNA using the deposited cDNA plasmid as a template. The polymerase chain reaction is carried out under routine conditions, for instance, in 25  $\mu$ l of reaction mixture with 0.5 ug of the above cDNA template. A convenient reaction mixture is 1.5-5 mM MgCl₂, 0.01% (w/v) gelatin, 20  $\mu$ M each of dATP, dCTP, dGTP, dTTP, 25 pmol of each primer and 0.25 Unit of Taq polymerase. Thirty five

cycles of PCR (denaturation at 94°C for 1 min; annealing at 55°C for 1 min; elongation at 72°C for 1 min) are performed with a Perkin-Elmer Cetus automated thermal cycler. The amplified product is analyzed by agarose gel electrophoresis and the DNA band with expected molecular weight is excised and purified. The PCR product is verified to be the selected sequence by subcloning and sequencing the DNA product.

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Several methods are available for the identification of the 5' or 3' non-coding portions of a gene which may not be present in the deposited clone. These methods include but are not limited to, filter probing, clone enrichment using specific probes, and protocols similar or identical to 5' and 3' "RACE" protocols which are well known in the art. For instance, a method similar to 5' RACE is available for generating the missing 5' end of a desired full-length transcript. (Fromont-Racine et al., Nucleic Acids Res. 21(7):1683-1684 (1993)).

Briefly, a specific RNA oligonucleotide is ligated to the 5' ends of a population of RNA presumably containing full-length gene RNA transcripts. A primer set containing a primer specific to the ligated RNA oligonucleotide and a primer specific to a known sequence of the gene of interest is used to PCR amplify the 5' portion of the desired full-length gene. This amplified product may then be sequenced and used to generate the full length gene.

This above method starts with total RNA isolated from the desired source, although poly-A+RNA can be used. The RNA preparation can then be treated with phosphatase if necessary to eliminate 5' phosphate groups on degraded or damaged RNA which may interfere with the later RNA ligase step. The phosphatase should then be inactivated and the RNA treated with tobacco acid pyrophosphatase in order to remove the cap structure present at the 5' ends of messenger RNAs. This reaction leaves a 5' phosphate group at the 5' end of the cap cleaved RNA which can then be ligated to an RNA oligonucleotide using T4 RNA ligase.

This modified RNA preparation is used as a template for first strand cDNA synthesis using a gene specific oligonucleotide. The first strand synthesis reaction is used as a template for PCR amplification of the desired 5' end using a primer specific to the ligated RNA oligonucleotide and a primer specific to the known sequence of the gene of interest. The resultant product is then sequenced and analyzed to confirm that the 5' end sequence belongs to the desired gene.

# Example 2: Isolation of Genomic Clones Corresponding to a Polynucleotide

A human genomic P1 library (Genomic Systems, Inc.) is screened by PCR using primers selected for the sequence corresponding to SEQ ID NO:X according to the method described in Example 1. (See also, Sambrook.)

#### Example 3: Tissue specific expression analysis

The Human Genome Sciences, Inc. (HGS) database is derived from sequencing tissue and/or disease specific cDNA libraries. Libraries generated from a particular tissue are selected

and the specific tissue expression pattern of EST groups or assembled contigs within these libraries is determined by comparison of the expression patterns of those groups or contigs within the entire database. ESTs and assembled contigs which show tissue specific expression are selected.

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The original clone from which the specific EST sequence was generated, or in the case of an assembled contig, the clone from which the 5' most EST sequence was generated, is obtained from the catalogued library of clones and the insert amplified by PCR using methods known in the art. The PCR product is denatured and then transferred in 96 or 384 well format to a nylon membrane (Schleicher and Scheull) generating an array filter of tissue specific clones. Housekeeping genes, maize genes, and known tissue specific genes are included on the filters. These targets can be used in signal normalization and to validate assay sensitivity. Additional targets are included to monitor probe length and specificity of hybridization.

Radioactively labeled hybridization probes are generated by first strand cDNA synthesis per the manufacturer's instructions (Life Technologies) from mRNA/RNA samples prepared from the specific tissue being analyzed (e.g., prostate, prostate cancer, ovarian, ovarian cancer, etc.). The hybridization probes are purified by gel exclusion chromatography, quantitated, and hybridized with the array filters in hybridization bottles at 65°C overnight. The filters are washed under stringent conditions and signals are captured using a Fuji phosphorimager.

Data is extracted using AIS software and following background subtraction, signal normalization is performed. This includes a normalization of filter-wide expression levels between different experimental runs. Genes that are differentially expressed in the tissue of interest are identified.

# Example 4: Chromosomal Mapping of the Polynucleotides

An oligonucleotide primer set is designed according to the sequence at the 5' end of SEQ ID NO:X. This primer preferably spans about 100 nucleotides. This primer set is then used in a polymerase chain reaction under the following set of conditions: 30 seconds, 95°C; 1 minute, 56°C; 1 minute, 70°C. This cycle is repeated 32 times followed by one 5 minute cycle at 70°C. Human, mouse, and hamster DNA is used as template in addition to a somatic cell hybrid panel containing individual chromosomes or chromosome fragments (Bios, Inc). The reactions are analyzed on either 8% polyacrylamide gels or 3.5 % agarose gels. Chromosome mapping is determined by the presence of an approximately 100 bp PCR fragment in the particular somatic cell hybrid.

#### Example 5: Bacterial Expression of a Polypeptide

A polynucleotide encoding a polypeptide of the present invention is amplified using PCR oligonucleotide primers corresponding to the 5' and 3' ends of the DNA sequence, as outlined in

Example 1, to synthesize insertion fragments. The primers used to amplify the cDNA insert should preferably contain restriction sites, such as BamHI and XbaI, at the 5' end of the primers in order to clone the amplified product into the expression vector. For example, BamHI and XbaI correspond to the restriction enzyme sites on the bacterial expression vector pQE-9. (Qiagen, Inc., Chatsworth, CA). This plasmid vector encodes antibiotic resistance (Ampr), a bacterial origin of replication (ori), an IPTG-regulatable promoter/operator (P/O), a ribosome binding site (RBS), a 6-histidine tag (6-His), and restriction enzyme cloning sites.

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The pQE-9 vector is digested with BamHI and XbaI and the amplified fragment is ligated into the pQE-9 vector maintaining the reading frame initiated at the bacterial RBS. The ligation mixture is then used to transform the E. coli strain M15/rep4 (Qiagen, Inc.) which contains multiple copies of the plasmid pREP4, which expresses the lacI repressor and also confers kanamycin resistance (Kan^r). Transformants are identified by their ability to grow on LB plates and ampicillin/kanamycin resistant colonies are selected. Plasmid DNA is isolated and confirmed by restriction analysis.

Clones containing the desired constructs are grown overnight (O/N) in liquid culture in LB media supplemented with both Amp (100 ug/ml) and Kan (25 ug/ml). The O/N culture is used to inoculate a large culture at a ratio of 1:100 to 1:250. The cells are grown to an optical density 600 (O.D.⁶⁰⁰) of between 0.4 and 0.6. IPTG (Isopropyl-B-D-thiogalacto pyranoside) is then added to a final concentration of 1 mM. IPTG induces by inactivating the lacI repressor, clearing the P/O leading to increased gene expression.

Cells are grown for an extra 3 to 4 hours. Cells are then harvested by centrifugation (20 mins at 6000Xg). The cell pellet is solubilized in the chaotropic agent 6 Molar Guanidine HCl by stirring for 3-4 hours at 4°C. The cell debris is removed by centrifugation, and the supernatant containing the polypeptide is loaded onto a nickel-nitrilo-tri-acetic acid ("Ni-NTA") affinity resin column (available from QIAGEN, Inc., supra). Proteins with a 6 x His tag bind to the Ni-NTA resin with high affinity and can be purified in a simple one-step procedure (for details see: The QIAexpressionist (1995) QIAGEN, Inc., supra).

Briefly, the supernatant is loaded onto the column in 6 M guanidine-HCl, pH 8. The column is first washed with 10 volumes of 6 M guanidine-HCl, pH 8, then washed with 10 volumes of 6 M guanidine-HCl pH 6, and finally the polypeptide is eluted with 6 M guanidine-HCl, pH 5.

The purified protein is then renatured by dialyzing it against phosphate-buffered saline (PBS) or 50 mM Na-acetate, pH 6 buffer plus 200 mM NaCl. Alternatively, the protein can be successfully refolded while immobilized on the Ni-NTA column. The recommended conditions are as follows: renature using a linear 6M-1M urea gradient in 500 mM NaCl, 20% glycerol, 20 mM Tris/HCl pH 7.4, containing protease inhibitors. The renaturation should be performed over a

period of 1.5 hours or more. After renaturation the proteins are eluted by the addition of 250 mM immidazole. Immidazole is removed by a final dialyzing step against PBS or 50 mM sodium acetate pH 6 buffer plus 200 mM NaCl. The purified protein is stored at 4°C or frozen at -80°C.

In addition to the above expression vector, the present invention further includes an expression vector, called pHE4a (ATCC Accession Number 209645, deposited on February 25, 1998) which contains phage operator and promoter elements operatively linked to a polynucleotide of the present invention, called pHE4a. (ATCC Accession Number 209645, deposited on February 25, 1998.) This vector contains: 1) a neomycinphosphotransferase gene as a selection marker, 2) an E. coli origin of replication, 3) a T5 phage promoter sequence, 4) two lac operator sequences, 5) a Shine-Delgarno sequence, and 6) the lactose operon repressor gene (lacIq). The origin of replication (oriC) is derived from pUC19 (LTI, Gaithersburg, MD). The promoter and operator sequences are made synthetically.

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DNA can be inserted into the pHE4a by restricting the vector with NdeI and XbaI, BamHI, XhoI, or Asp718, running the restricted product on a gel, and isolating the larger fragment (the stuffer fragment should be about 310 base pairs). The DNA insert is generated according to the PCR protocol described in Example 1, using PCR primers having restriction sites for NdeI (5' primer) and XbaI, BamHI, XhoI, or Asp718 (3' primer). The PCR insert is gel purified and restricted with compatible enzymes. The insert and vector are ligated according to standard protocols.

The engineered vector could easily be substituted in the above protocol to express protein in a bacterial system.

### Example 6: Purification of a Polypeptide from an Inclusion Body

The following alternative method can be used to purify a polypeptide expressed in *E coli* when it is present in the form of inclusion bodies. Unless otherwise specified, all of the following steps are conducted at 4-10°C.

Upon completion of the production phase of the *E. coli* fermentation, the cell culture is cooled to 4-10°C and the cells harvested by continuous centrifugation at 15,000 rpm (Heraeus Sepatech). On the basis of the expected yield of protein per unit weight of cell paste and the amount of purified protein required, an appropriate amount of cell paste, by weight, is suspended in a buffer solution containing 100 mM Tris, 50 mM EDTA, pH 7.4. The cells are dispersed to a homogeneous suspension using a high shear mixer.

The cells are then lysed by passing the solution through a microfluidizer (Microfuidics, Corp. or APV Gaulin, Inc.) twice at 4000-6000 psi. The homogenate is then mixed with NaCl solution to a final concentration of 0.5 M NaCl, followed by centrifugation at 7000 xg for 15 min. The resultant pellet is washed again using 0.5M NaCl, 100 mM Tris, 50 mM EDTA, pH 7.4.

The resulting washed inclusion bodies are solubilized with 1.5 M guanidine hydrochloride (GuHCl) for 2-4 hours. After 7000 xg centrifugation for 15 min., the pellet is discarded and the polypeptide containing supernatant is incubated at 4°C overnight to allow further GuHCl extraction.

Following high speed centrifugation (30,000 xg) to remove insoluble particles, the GuHCl solubilized protein is refolded by quickly mixing the GuHCl extract with 20 volumes of buffer containing 50 mM sodium, pH 4.5, 150 mM NaCl, 2 mM EDTA by vigorous stirring. The refolded diluted protein solution is kept at 4°C without mixing for 12 hours prior to further purification steps.

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To clarify the refolded polypeptide solution, a previously prepared tangential filtration unit equipped with 0.16 µm membrane filter with appropriate surface area (e.g., Filtron), equilibrated with 40 mM sodium acetate, pH 6.0 is employed. The filtered sample is loaded onto a cation exchange resin (e.g., Poros HS-50, Perseptive Biosystems). The column is washed with 40 mM sodium acetate, pH 6.0 and eluted with 250 mM, 500 mM, 1000 mM, and 1500 mM NaCl in the same buffer, in a stepwise manner. The absorbance at 280 nm of the effluent is continuously monitored. Fractions are collected and further analyzed by SDS-PAGE.

Fractions containing the polypeptide are then pooled and mixed with 4 volumes of water. The diluted sample is then loaded onto a previously prepared set of tandem columns of strong anion (Poros HQ-50, Perseptive Biosystems) and weak anion (Poros CM-20, Perseptive Biosystems) exchange resins. The columns are equilibrated with 40 mM sodium acetate, pH 6.0. Both columns are washed with 40 mM sodium acetate, pH 6.0, 200 mM NaCl. The CM-20 column is then eluted using a 10 column volume linear gradient ranging from 0.2 M NaCl, 50 mM sodium acetate, pH 6.0. Fractions are collected under constant A₂₈₀ monitoring of the effluent. Fractions containing the polypeptide (determined, for instance, by 16% SDS-PAGE) are then pooled.

The resultant polypeptide should exhibit greater than 95% purity after the above refolding and purification steps. No major contaminant bands should be observed from Commassie blue stained 16% SDS-PAGE gel when 5 µg of purified protein is loaded. The purified protein can also be tested for endotoxin/LPS contamination, and typically the LPS content is less than 0.1 ng/ml according to LAL assays.

# Example 7: Cloning and Expression of a Polypeptide in a Baculovirus Expression System

In this example, the plasmid shuttle vector pA2 is used to insert a polynucleotide into a baculovirus to express a polypeptide. This expression vector contains the strong polyhedrin promoter of the *Autographa californica* nuclear polyhedrosis virus (AcMNPV) followed by convenient restriction sites such as BamHI, Xba I and Asp718. The polyadenylation site of the

simian virus 40 ("SV40") is used for efficient polyadenylation. For easy selection of recombinant virus, the plasmid contains the beta-galactosidase gene from *E. coli* under control of a weak Drosophila promoter in the same orientation, followed by the polyadenylation signal of the polyhedrin gene. The inserted genes are flanked on both sides by viral sequences for cell-mediated homologous recombination with wild-type viral DNA to generate a viable virus that express the cloned polynucleotide.

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Many other baculovirus vectors can be used in place of the vector above, such as pAc373, pVL941, and pAcIM1, as one skilled in the art would readily appreciate, as long as the construct provides appropriately located signals for transcription, translation, secretion and the like, including a signal peptide and an in-frame AUG as required. Such vectors are described, for instance, in Luckow et al., Virology 170:31-39 (1989).

Specifically, the cDNA sequence contained in the deposited clone, including the AUG initiation codon, is amplified using the PCR protocol described in Example 1. If a naturally occurring signal sequence is used to produce the polypeptide of the present invention, the pA2 vector does not need a second signal peptide. Alternatively, the vector can be modified (pA2 GP) to include a baculovirus leader sequence, using the standard methods described in Summers et al., "A Manual of Methods for Baculovirus Vectors and Insect Cell Culture Procedures," Texas Agricultural Experimental Station Bulletin No. 1555 (1987).

The amplified fragment is isolated from a 1% agarose gel using a commercially available kit ("Geneclean," BIO 101 Inc., La Jolla, Ca.). The fragment then is digested with appropriate restriction enzymes and again purified on a 1% agarose gel.

The plasmid is digested with the corresponding restriction enzymes and optionally, can be dephosphorylated using calf intestinal phosphatase, using routine procedures known in the art. The DNA is then isolated from a 1% agarose gel using a commercially available kit ("Geneclean" BIO 101 Inc., La Jolla, Ca.).

The fragment and the dephosphorylated plasmid are ligated together with T4 DNA ligase. E. coli HB101 or other suitable E. coli hosts such as XL-1 Blue (Stratagene Cloning Systems, La Jolla, CA) cells are transformed with the ligation mixture and spread on culture plates. Bacteria containing the plasmid are identified by digesting DNA from individual colonies and analyzing the digestion product by gel electrophoresis. The sequence of the cloned fragment is confirmed by DNA sequencing.

Five  $\mu g$  of a plasmid containing the polynucleotide is co-transfected with 1.0  $\mu g$  of a commercially available linearized baculovirus DNA ("BaculoGoldTM baculovirus DNA, Pharmingen, San Diego, CA), using the lipofection method described by Felgner et al., Proc. Natl. Acad. Sci. USA 84:7413-7417 (1987). One  $\mu g$  of BaculoGoldTM virus DNA and 5  $\mu g$  of the plasmid are mixed in a sterile well of a microtiter plate containing 50  $\mu l$  of serum-free Grace's medium (Life Technologies Inc., Gaithersburg, MD). Afterwards, 10  $\mu l$  Lipofectin plus 90  $\mu l$ 

Grace's medium are added, mixed and incubated for 15 minutes at room temperature. Then the transfection mixture is added drop-wise to Sf9 insect cells (ATCC CRL 1711) seeded in a 35 mm tissue culture plate with 1 ml Grace's medium without serum. The plate is then incubated for 5 hours at 27° C. The transfection solution is then removed from the plate and 1 ml of Grace's insect medium supplemented with 10% fetal calf serum is added. Cultivation is then continued at 27° C for four days.

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After four days the supernatant is collected and a plaque assay is performed, as described by Summers and Smith, *supra*. An agarose gel with "Blue Gal" (Life Technologies Inc., Gaithersburg) is used to allow easy identification and isolation of gal-expressing clones, which produce blue-stained plaques. (A detailed description of a "plaque assay" of this type can also be found in the user's guide for insect cell culture and baculovirology distributed by Life Technologies Inc., Gaithersburg, page 9-10.) After appropriate incubation, blue stained plaques are picked with the tip of a micropipettor (e.g., Eppendorf). The agar containing the recombinant viruses is then resuspended in a microcentrifuge tube containing  $200 \mu l$  of Grace's medium and the suspension containing the recombinant baculovirus is used to infect Sf9 cells seeded in 35 mm dishes. Four days later the supernatants of these culture dishes are harvested and then they are stored at 4° C.

To verify the expression of the polypeptide, Sf9 cells are grown in Grace's medium supplemented with 10% heat-inactivated FBS. The cells are infected with the recombinant baculovirus containing the polynucleotide at a multiplicity of infection ("MOI") of about 2. If radiolabeled proteins are desired, 6 hours later the medium is removed and is replaced with SF900 II medium minus methionine and cysteine (available from Life Technologies Inc., Rockville, MD). After 42 hours, 5  $\mu$ Ci of ³⁵S-methionine and 5  $\mu$ Ci ³⁵S-cysteine (available from Amersham) are added. The cells are further incubated for 16 hours and then are harvested by centrifugation. The proteins in the supernatant as well as the intracellular proteins are analyzed by SDS-PAGE followed by autoradiography (if radiolabeled).

Microsequencing of the amino acid sequence of the amino terminus of purified protein may be used to determine the amino terminal sequence of the produced protein.

#### Example 8: Expression of a Polypeptide in Mammalian Cells

The polypeptide of the present invention can be expressed in a mammalian cell. A typical mammalian expression vector contains a promoter element, which mediates the initiation of transcription of mRNA, a protein coding sequence, and signals required for the termination of transcription and polyadenylation of the transcript. Additional elements include enhancers, Kozak sequences and intervening sequences flanked by donor and acceptor sites for RNA splicing. Highly efficient transcription is achieved with the early and late promoters from SV40, the long

terminal repeats (LTRs) from Retroviruses, e.g., RSV, HTLVI, HIVI and the early promoter of the cytomegalovirus (CMV). However, cellular elements can also be used (e.g., the human actin promoter).

Suitable expression vectors for use in practicing the present invention include, for example, vectors such as pSVL and pMSG (Pharmacia, Uppsala, Sweden), pRSVcat (ATCC 37152), pSV2dhfr (ATCC 37146), pBC12MI (ATCC 67109), pCMVSport 2.0, and pCMVSport 3.0. Mammalian host cells that could be used include, human Hela, 293, H9 and Jurkat cells, mouse NIH3T3 and C127 cells, Cos 1, Cos 7 and CV1, quail QC1-3 cells, mouse L cells and Chinese hamster ovary (CHO) cells.

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Alternatively, the polypeptide can be expressed in stable cell lines containing the polynucleotide integrated into a chromosome. The co-transfection with a selectable marker such as DHFR, gpt, neomycin, or hygromycin allows the identification and isolation of the transfected cells.

The transfected gene can also be amplified to express large amounts of the encoded protein. The DHFR (dihydrofolate reductase) marker is useful in developing cell lines that carry several hundred or even several thousand copies of the gene of interest. (See, e.g., Alt, F. W., et al., J. Biol. Chem. 253:1357-1370 (1978); Hamlin, J. L. and Ma, C., Biochem. et Biophys. Acta, 1097:107-143 (1990); Page, M. J. and Sydenham, M. A., Biotechnology 9:64-68 (1991)). Another useful selection marker is the enzyme glutamine synthase (GS) (Murphy et al., Biochem J. 227:277-279 (1991); Bebbington et al., Bio/Technology 10:169-175 (1992). Using these markers, the mammalian cells are grown in selective medium and the cells with the highest resistance are selected. These cell lines contain the amplified gene(s) integrated into a chromosome. Chinese hamster ovary (CHO) and NSO cells are often used for the production of proteins.

Derivatives of the plasmid pSV2-dhfr (ATCC Accession No. 37146), the expression vectors pC4 (ATCC Accession No. 209646) and pC6 (ATCC Accession No.209647) contain the strong promoter (LTR) of the Rous Sarcoma Virus (Cullen et al., Molecular and Cellular Biology, 438-447 (March, 1985)) plus a fragment of the CMV-enhancer (Boshart et al., Cell 41:521-530 (1985)). Multiple cloning sites, e.g., with the restriction enzyme cleavage sites BamHI, XbaI and Asp718, facilitate the cloning of the gene of interest. The vectors also contain the 3' intron, the polyadenylation and termination signal of the rat preproinsulin gene, and the mouse DHFR gene under control of the SV40 early promoter.

Specifically, the plasmid pC6, for example, is digested with appropriate restriction enzymes and then dephosphorylated using calf intestinal phosphates by procedures known in the art. The vector is then isolated from a 1% agarose gel.

A polynucleotide of the present invention is amplified according to the protocol outlined in Example 1. If a naturally occurring signal sequence is used to produce the polypeptide of the

present invention, the vector does not need a second signal peptide. Alternatively, if a naturally occurring signal sequence is not used, the vector can be modified to include a heterologous signal sequence. (See, e.g., International Publication No. WO 96/34891.)

The amplified fragment is isolated from a 1% agarose gel using a commercially available kit ("Geneclean," BIO 101 Inc., La Jolla, Ca.). The fragment then is digested with appropriate restriction enzymes and again purified on a 1% agarose gel.

The amplified fragment is then digested with the same restriction enzyme and purified on a 1% agarose gel. The isolated fragment and the dephosphorylated vector are then ligated with T4 DNA ligase. E. coli HB101 or XL-1 Blue cells are then transformed and bacteria are identified that contain the fragment inserted into plasmid pC6 using, for instance, restriction enzyme analysis.

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Chinese hamster ovary cells lacking an active DHFR gene is used for transfection. Five  $\mu$ g of the expression plasmid pC6 or pC4 is cotransfected with 0.5  $\mu$ g of the plasmid pSVneo using lipofectin (Felgner et al., supra). The plasmid pSV2-neo contains a dominant selectable marker, the neo gene from Tn5 encoding an enzyme that confers resistance to a group of antibiotics including G418. The cells are seeded in alpha minus MEM supplemented with 1 mg/ml G418. After 2 days, the cells are trypsinized and seeded in hybridoma cloning plates (Greiner, Germany) in alpha minus MEM supplemented with 10, 25, or 50 ng/ml of methotrexate plus 1 mg/ml G418. After about 10-14 days single clones are trypsinized and then seeded in 6-well petri dishes or 10 ml flasks using different concentrations of methotrexate (50 nM, 100 nM, 200 nM, 400 nM, 800 nM). Clones growing at the highest concentrations of methotrexate are then transferred to new 6-well plates containing even higher concentrations of methotrexate (1  $\mu$ M, 2  $\mu$ M, 5  $\mu$ M, 10 mM, 20 mM). The same procedure is repeated until clones are obtained which grow at a concentration of 100 - 200  $\mu$ M. Expression of the desired gene product is analyzed, for instance, by SDS-PAGE and Western blot or by reversed phase HPLC analysis.

## Example 9: Protein Fusions

The polypeptides of the present invention are preferably fused to other proteins. These fusion proteins can be used for a variety of applications. For example, fusion of the present polypeptides to His-tag, HA-tag, protein A, IgG domains, and maltose binding protein facilitates purification. (See Example 5; see also EP A 394,827; Traunecker, et al., Nature 331:84-86 (1988)). Similarly, fusion to IgG-1, IgG-3, and albumin increases the halflife time *in vivo*. Nuclear localization signals fused to the polypeptides of the present invention can target the protein to a specific subcellular localization, while covalent heterodimer or homodimers can increase or decrease the activity of a fusion protein. Fusion proteins can also create chimeric molecules having more than one function. Finally, fusion proteins can increase solubility and/or stability of the fused protein compared to the non-fused protein. All of the types of fusion proteins described

above can be made by modifying the following protocol, which outlines the fusion of a polypeptide to an IgG molecule, or the protocol described in Example 5.

Briefly, the human Fc portion of the IgG molecule can be PCR amplified, using primers that span the 5' and 3' ends of the sequence described below. These primers also should have convenient restriction enzyme sites that will facilitate cloning into an expression vector, preferably a mammalian expression vector.

For example, if pC4 (ATCC Accession No. 209646) is used, the human Fc portion can be ligated into the BamHI cloning site. Note that the 3' BamHI site should be destroyed. Next, the vector containing the human Fc portion is re-restricted with BamHI, linearizing the vector, and a polynucleotide of the present invention, isolated by the PCR protocol described in Example 1, is ligated into this BamHI site. Note that the polynucleotide is cloned without a stop codon, otherwise a fusion protein will not be produced.

If the naturally occurring signal sequence is used to produce the polypeptide of the present invention, pC4 does not need a second signal peptide. Alternatively, if the naturally occurring signal sequence is not used, the vector can be modified to include a heterologous signal sequence. (See, e.g., International Publication No. WO 96/34891.)

## Human IgG Fc region:

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GGGATCCGGAGCCCAAATCTTCTGACAAAACTCACACATGCCCACCGTGCCCAGCAC
CTGAATTCGAGGGTGCACCGTCAGTCTTCCTCTTCCCCCCAAAACCCAAGGACACCCT
CATGATCTCCCGGACTCCTGAGGTCACATGCGTGGTGGTGGACGTAAGCCACGAAGA
CCCTGAGGTCAAGTTCAACTGGTACGTGGACGCGTGGAGGTGCATAATGCCAAGAC
AAAGCCGCGGGAGGAGCAGTACAACAGCACGTACCGTGGTCAGCGTCCTCACCGT
CCTGCACCAGGACTGCTGAATGGCAAGGAGTACAAGTGCAAGGTCTCCAACAAAGC
CCTCCCAACCCCCATCGAGAAAACCATCTCCAAAGCCAAAGGGCAGCCCCGAGAACC
ACAGGTGTACACCTGCCCCCATCCCGGGATGAGCTGACCAAGAACCAGGTCAGCCT
GACCTGCCTGGTCAAAGGCTTCTATCCAAGCGACATCGCCGTGGAGTGGGAGAGCAA
TGGGCAGCCGGAGAACAACTACAAGACCACGCCTCCCGTGGACTCCGACGGCTC
CTTCTTCCTCTACAGCAAGCTCACCGTGGACAAGAGCAGGGGAACGT
CTTCTTCCTCTACAGCAAGTCACCGTGGACAACCACTACACGCAGAAGAGCCTC
TCCCTGTCTCCGGGTAAATGAGTGCGACGGCCGCGACTCTAGAGGAT (SEQ ID NO: 1)

# Example 10: Production of an Antibody from a Polypeptide

#### a) Hybridoma Technology

The antibodies of the present invention can be prepared by a variety of methods. (See, Current Protocols, Chapter 2.) As one example of such methods, cells expressing a polypeptide of the present invention are administered to an animal to induce the production of sera containing

polyclonal antibodies. In a preferred method, a preparation of a polypeptide of the present invention is prepared and purified to render it substantially free of natural contaminants. Such a preparation is then introduced into an animal in order to produce polyclonal antisera of greater specific activity.

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Monoclonal antibodies specific for a polypeptide of the present invention are prepared using hybridoma technology (Kohler et al., Nature 256:495 (1975); Kohler et al., Eur. J. Immunol. 6:511 (1976); Kohler et al., Eur. J. Immunol. 6:292 (1976); Hammerling et al., in: Monoclonal Antibodies and T-Cell Hybridomas, Elsevier, N.Y., pp. 563-681 (1981)). In general, an animal (preferably a mouse) is immunized with a polypeptide of the present invention or, more preferably, with a secreted polypeptide-expressing cell. Such polypeptide-expressing cells are cultured in any suitable tissue culture medium, preferably in Earle's modified Eagle's medium supplemented with 10% fetal bovine serum (inactivated at about 56°C), and supplemented with about 10 g/l of nonessential amino acids, about 1,000 U/ml of penicillin, and about 100  $\mu$ g/ml of streptomycin.

The splenocytes of such mice are extracted and fused with a suitable myeloma cell line. Any suitable myeloma cell line may be employed in accordance with the present invention; however, it is preferable to employ the parent myeloma cell line (SP2O), available from the ATCC. After fusion, the resulting hybridoma cells are selectively maintained in HAT medium, and then cloned by limiting dilution as described by Wands et al. (Gastroenterology 80:225-232 (1981)). The hybridoma cells obtained through such a selection are then assayed to identify clones which secrete antibodies capable of binding the polypeptide of the present invention.

Alternatively, additional antibodies capable of binding to a polypeptide of the present invention can be produced in a two-step procedure using anti-idiotypic antibodies. Such a method makes use of the fact that antibodies are themselves antigens, and therefore, it is possible to obtain an antibody which binds to a second antibody. In accordance with this method, protein specific antibodies are used to immunize an animal, preferably a mouse. The splenocytes of such an animal are then used to produce hybridoma cells, and the hybridoma cells are screened to identify clones which produce an antibody whose ability to bind to the polypeptide-specific antibody can be blocked by said polypeptide. Such antibodies comprise anti-idiotypic antibodies to the polypeptide-specific antibody and are used to immunize an animal to induce formation of further polypeptide-specific antibodies.

For *in vivo* use of antibodies in humans, an antibody is "humanized". Such antibodies can be produced using genetic constructs derived from hybridoma cells producing the monoclonal antibodies described above. Methods for producing chimeric and humanized antibodies are known in the art and are discussed herein. (See, for review, Morrison, Science 229:1202 (1985); Oi et al., BioTechniques 4:214 (1986); Cabilly et al., U.S. Patent No. 4,816,567; Taniguchi et al., EP 171496; Morrison et al., EP 173494; Neuberger et al., WO 8601533; Robinson et al., International

Publication No. WO 8702671; Boulianne et al., Nature 312:643 (1984); Neuberger et al., Nature 314:268 (1985)).

b) Isolation Of Antibody Fragments Directed Against a Polypeptide of the Present Invention From A Library Of scFvs

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Naturally occurring V-genes isolated from human PBLs are constructed into a library of antibody fragments which contain reactivities against a polypeptide of the present invention to which the donor may or may not have been exposed (see e.g., U.S. Patent 5,885,793 incorporated herein by reference in its entirety).

Rescue of the Library. A library of scFvs is constructed from the RNA of human PBLs as described in International Publication No. WO 92/01047. To rescue phage displaying antibody fragments, approximately  $10^9$  E. coli harboring the phagemid are used to inoculate 50 ml of 2xTY containing 1% glucose and  $100 \mu g/ml$  of ampicillin (2xTY-AMP-GLU) and grown to an O.D. of 0.8 with shaking. Five ml of this culture is used to inoculate 50 ml of 2xTY-AMP-GLU, 2 x 108 TU of delta gene 3 helper (M13 delta gene III, see International Publication No. WO 92/01047) are added and the culture incubated at 37°C for 45 minutes without shaking and then at 37°C for 45 minutes with shaking. The culture is centrifuged at 4000 r.p.m. for 10 min. and the pellet resuspended in 2 liters of 2xTY containing  $100 \mu g/ml$  ampicillin and 50 ug/ml kanamycin and grown overnight. Phage are prepared as described in International Publication No. WO 92/01047.

M13 delta gene III is prepared as follows: M13 delta gene III helper phage does not encode gene III protein, hence the phage(mid) displaying antibody fragments have a greater avidity of binding to antigen. Infectious M13 delta gene III particles are made by growing the helper phage in cells harboring a pUC19 derivative supplying the wild type gene III protein during phage morphogenesis. The culture is incubated for 1 hour at 37° C without shaking and then for a further hour at 37°C with shaking. Cells are spun down (IEC-Centra 8,400 r.p.m. for 10 min), resuspended in 300 ml 2xTY broth containing 100  $\mu$ g ampicillin/ml and 25  $\mu$ g kanamycin/ml (2xTY-AMP-KAN) and grown overnight, shaking at 37°C. Phage particles are purified and concentrated from the culture medium by two PEG-precipitations (Sambrook et al., 1990), resuspended in 2 ml PBS and passed through a 0.45  $\mu$ m filter (Minisart NML; Sartorius) to give a final concentration of approximately  $10^{13}$  transducing units/ml (ampicillin-resistant clones).

Panning of the Library. Immunotubes (Nunc) are coated overnight in PBS with 4 ml of either 100  $\mu$ g/ml or 10  $\mu$ g/ml of a polypeptide of the present invention. Tubes are blocked with 2% Marvel-PBS for 2 hours at 37°C and then washed 3 times in PBS. Approximately  $10^{13}$  TU of phage is applied to the tube and incubated for 30 minutes at room temperature tumbling on an over and under turntable and then left to stand for another 1.5 hours. Tubes are washed 10 times with PBS 0.1% Tween-20 and 10 times with PBS. Phage are eluted by adding 1 ml of 100 mM triethylamine and rotating 15 minutes on an under and over turntable after which the solution is

immediately neutralized with 0.5 ml of 1.0M Tris-HCl, pH 7.4. Phage are then used to infect 10 ml of mid-log E. coli TG1 by incubating eluted phage with bacteria for 30 minutes at 37°C. The E. coli are then plated on TYE plates containing 1% glucose and 100  $\mu$ g/ml ampicillin. The resulting bacterial library is then rescued with delta gene 3 helper phage as described above to prepare phage for a subsequent round of selection. This process is then repeated for a total of 4 rounds of affinity purification with tube-washing increased to 20 times with PBS, 0.1% Tween-20 and 20 times with PBS for rounds 3 and 4.

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Characterization of Binders. Eluted phage from the 3rd and 4th rounds of selection are used to infect E. coli HB 2151 and soluble scFv is produced (Marks, et al., 1991) from single colonies for assay. ELISAs are performed with microtitre plates coated with either 10 pg/ml of the polypeptide of the present invention in 50 mM bicarbonate pH 9.6. Clones positive in ELISA are further characterized by PCR fingerprinting (see, e.g., International Publication No. WO 92/01047) and then by sequencing. These ELISA positive clones may also be further characterized by techniques known in the art, such as, for example, epitope mapping, binding affinity, receptor signal transduction, ability to block or competitively inhibit antibody/antigen binding, and competitive agonistic or antagonistic activity.

# Example 11: Method of Determining Alterations in a Gene Corresponding to a Polynucleotide

RNA isolated from entire families or individual patients presenting with a hematopoietic or hematologic disease or disorder is isolated. cDNA is then generated from these RNA samples using protocols known in the art. (See, Sambrook.) The cDNA is then used as a template for PCR, employing primers surrounding regions of interest in SEQ ID NO:X; and/or the nucleotide sequence of the cDNA contained in ATCC Deposit No:Z. Suggested PCR conditions consist of 35 cycles at 95 degrees C for 30 seconds; 60-120 seconds at 52-58 degrees C; and 60-120 seconds at 70 degrees C, using buffer solutions described in Sidransky et al., Science 252:706 (1991).

PCR products are then sequenced using primers labeled at their 5' end with T4 polynucleotide kinase, employing SequiTherm Polymerase (Epicentre Technologies). The intronexon boundaries of selected exons is also determined and genomic PCR products analyzed to confirm the results. PCR products harboring suspected mutations are then cloned and sequenced to validate the results of the direct sequencing.

PCR products are cloned into T-tailed vectors as described in Holton et al., Nucleic Acids Research, 19:1156 (1991) and sequenced with T7 polymerase (United States Biochemical). Affected individuals are identified by mutations not present in unaffected individuals.

Genomic rearrangements are also observed as a method of determining alterations in a gene corresponding to a polynucleotide. Genomic clones isolated according to Example 2 are nick-translated with digoxigenindeoxy-uridine 5'-triphosphate (Boehringer Manheim), and FISH performed as described in Johnson et al., Methods Cell Biol. 35:73-99 (1991). Hybridization with

the labeled probe is carried out using a vast excess of human cot-1 DNA for specific hybridization to the corresponding genomic locus.

Chromosomes are counterstained with 4,6-diamino-2-phenylidole and propidium iodide, producing a combination of C- and R-bands. Aligned images for precise mapping are obtained using a triple-band filter set (Chroma Technology, Brattleboro, VT) in combination with a cooled charge-coupled device camera (Photometrics, Tucson, AZ) and variable excitation wavelength filters. (Johnson et al., Genet. Anal. Tech. Appl., 8:75 (1991)). Image collection, analysis and chromosomal fractional length measurements are performed using the ISee Graphical Program System. (Inovision Corporation, Durham, NC.) Chromosome alterations of the genomic region hybridized by the probe are identified as insertions, deletions, and translocations. These alterations are used as a diagnostic marker for an associated disease.

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# Example 12: Method of Detecting Abnormal Levels of a Polypeptide in a Biological Sample

A polypeptide of the present invention can be detected in a biological sample, and if an increased or decreased level of the polypeptide is detected, this polypeptide is a marker for a particular phenotype. Methods of detection are numerous, and thus, it is understood that one skilled in the art can modify the following assay to fit their particular needs.

For example, antibody-sandwich ELISAs are used to detect polypeptides in a sample, preferably a biological sample. Wells of a microtiter plate are coated with specific antibodies, at a final concentration of 0.2 to 10 ug/ml. The antibodies are either monoclonal or polyclonal and are produced by the method described in Example 10. The wells are blocked so that non-specific binding of the polypeptide to the well is reduced.

The coated wells are then incubated for > 2 hours at RT with a sample containing the polypeptide. Preferably, serial dilutions of the sample should be used to validate results. The plates are then washed three times with deionized or distilled water to remove unbound polypeptide.

Next, 50 ul of specific antibody-alkaline phosphatase conjugate, at a concentration of 25-400 ng, is added and incubated for 2 hours at room temperature. The plates are again washed three times with deionized or distilled water to remove unbound conjugate.

Add 75 ul of 4-methylumbelliferyl phosphate (MUP) or p-nitrophenyl phosphate (NPP) substrate solution to each well and incubate 1 hour at room temperature. Measure the reaction by a microtiter plate reader. Prepare a standard curve, using serial dilutions of a control sample, and plot polypeptide concentration on the X-axis (log scale) and fluorescence or absorbance of the Y-axis (linear scale). Interpolate the concentration of the polypeptide in the sample using the standard curve.

#### Example 13: Formulation

The invention also provides methods of preventing, treating and/or ameliorating a hematopoietic and hematologic disease or disorder by administration to a subject of an effective amount of a Therapeutic. By therapeutic is meant polynucleotides or polypeptides of the invention (including fragments and variants), agonists or antagonists thereof, and/or antibodies thereto, in combination with a pharmaceutically acceptable carrier type (e.g., a sterile carrier).

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The Therapeutic will be formulated and dosed in a fashion consistent with good medical practice, taking into account the clinical condition of the individual patient (especially the side effects of treatment with the Therapeutic alone), the site of delivery, the method of administration, the scheduling of administration, and other factors known to practitioners. The "effective amount" for purposes herein is thus determined by such considerations.

As a general proposition, the total pharmaceutically effective amount of the Therapeutic administered parenterally per dose will be in the range of about lug/kg/day to 10 mg/kg/day of patient body weight, although, as noted above, this will be subject to therapeutic discretion. More preferably, this dose is at least 0.01 mg/kg/day, and most preferably for humans between about 0.01 and 1 mg/kg/day for the hormone. If given continuously, the Therapeutic is typically administered at a dose rate of about 1 ug/kg/hour to about 50 ug/kg/hour, either by 1-4 injections per day or by continuous subcutaneous infusions, for example, using a mini-pump. An intravenous bag solution may also be employed. The length of treatment needed to observe changes and the interval following treatment for responses to occur appears to vary depending on the desired effect.

Therapeutics can be are administered orally, rectally, parenterally, intracistemally, intravaginally, intraperitoneally, topically (as by powders, ointments, gels, drops or transdermal patch), bucally, or as an oral or nasal spray. "Pharmaceutically acceptable carrier" refers to a nontoxic solid, semisolid or liquid filler, diluent, encapsulating material or formulation auxiliary of any. The term "parenteral" as used herein refers to modes of administration which include intravenous, intramuscular, intraperitoneal, intrasternal, subcutaneous and intraarticular injection and infusion.

Therapeutics of the invention are also suitably administered by sustained-release systems. Suitable examples of sustained-release Therapeutics are administered orally, rectally, parenterally, intracistemally, intravaginally, intraperitoneally, topically (as by powders, ointments, gels, drops or transdermal patch), bucally, or as an oral or nasal spray. "Pharmaceutically acceptable carrier" refers to a non-toxic solid, semisolid or liquid filler, diluent, encapsulating material or formulation auxiliary of any type. The term "parenteral" as used herein refers to modes of administration which include intravenous, intramuscular, intraperitoneal, intrasternal, subcutaneous and intraarticular injection and infusion.

Therapeutics of the invention are also suitably administered by sustained-release systems. Suitable examples of sustained-release Therapeutics include suitable polymeric materials (such as, for example, semi-permeable polymer matrices in the form of shaped articles, e.g., films, or mirocapsules), suitable hydrophobic materials (for example as an emulsion in an acceptable oil) or ion exchange resins, and sparingly soluble derivatives (such as, for example, a sparingly soluble salt).

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Sustained-release matrices include polylactides (U.S. Pat. No. 3,773,919, EP 58,481), copolymers of L-glutamic acid and gamma-ethyl-L-glutamate (Sidman et al., Biopolymers 22:547-556 (1983)), poly (2- hydroxyethyl methacrylate) (Langer et al., J. Biomed. Mater. Res. 15:167-277 (1981), and Langer, Chem. Tech. 12:98-105 (1982)), ethylene vinyl acetate (Langer et al., Id.) or poly-D-(-)-3-hydroxybutyric acid (EP 133,988).

In a preferred embodiment, polypeptide, polynucleotide, and antibody compositions of the invention are formulated in a biodegradable, polymeric drug delivery system, for example as described in U.S. Patent Nos. 4,938,763; 5,278,201; 5,278,202; 5,324,519; 5,340,849; and 5,487,897 and in International Publication Numbers WO01/35929, WO00/24374, and WO00/06117 which are hereby incorporated by reference in their entirety. In specific preferred embodiments the polypeptide, polynucleotide, and antibody compositions of the invention are formulated using the ATRIGEL® Biodegradable System of Atrix Laboratories, Inc. (Fort Collins, Colorado).

Examples of biodegradable polymers which can be used in the formulation of polypeptide, polynucleotide, and antibody compositions, include but are not limited to, polylactides, polyglycolides, polycaprolactones, polyamhydrides, polyamides, polyurethanes, polyesteramides, polyorthoesters, polydioxanones, polyacetals, polyketals, polycarbonates, polyorthocarbonates, polyhydroxybutyrates, polyhydroxyvalerates, polyphosphazenes, polyalkylene oxalates. polyalkylene succinates, poly(malic acid), poly(amino acids), poly(methyl vinyl ether), poly(maleic anhydride), polyvinylpyrrolidone, polyethylene glycol, polyhydroxycellulose, chitin, chitosan, and copolymers, terpolymers, or combinations or mixtures of the above materials. The preferred polymers are those that have a lower degree of crystallization and are more hydrophobic. These polymers and copolymers are more soluble in the biocompatible solvents than the highly crystalline polymers such as polyglycolide and chitin which also have a high degree of hydrogenbonding. Preferred materials with the desired solubility parameters are the polylactides, polycaprolactones, and copolymers of these with glycolide in which there are more amorphous regions to enhance solubility. In specific preferred embodiments, the biodegradable polymers which can be used in the formulation of polypeptide, polynucleotide, and antibody compositions are poly(lactide-co-glycolides). Polymer properties such as molecular weight, hydrophobicity, and lactide/glycolide ratio may be modified to obtain the desired polypeptide, polynucleotide, or

antibody release profile (See, e.g., Ravivarapu et al., Journal of Pharmaceutical Sciences 89:732-741 (2000), which is hereby incorporated by reference in its entirety).

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It is also preferred that the solvent for the biodegradable polymer be non-toxic, water miscible, and otherwise biocompatible. Examples of such solvents include, but are not limited to, N-methyl-2-pyrrolidone, 2-pyrrolidone, C2 to C6 alkanols, C1 to C15 alchohols, dils, triols, and tetraols such as ethanol, glycerine propylene glycol, butanol; C3 to C15 alkyl ketones such as acetone, diethyl ketone and methyl ethyl ketone; C3 to C15 esters such as methyl acetate, ethyl acetate, ethyl lactate; alkyl ketones such as methyl ethyl ketone, C1 to C15 amides such as dimethylformamide, dimethylacetamide and caprolactam; C3 to C20 ethers such as tetrahydrofuran, or solketal; tweens, triacetin, propylene carbonate, decylmethylsulfoxide, dimethyl sulfoxide, oleic acid, 1-dodecylazacycloheptan-2-one, Other preferred solvents are benzyl alchohol, benzyl benzoate, dipropylene glycol, tributyrin, ethyl oleate, glycerin, glycofural, isopropyl myristate, isopropyl palmitate, oleic acid, polyethylene glycol, propylene carbonate, and triethyl citrate. The most preferred solvents are N-methyl-2-pyrrolidone, 2-pyrrolidone, dimethyl sulfoxide, triacetin, and propylene carbonate because of the solvating ability and their compatibility.

Additionally, formulations comprising polypeptide, polynucleotide, and antibody compositions and a biodegradable polymer may also include release-rate modification agents and/or pore-forming agents. Examples of release-rate modification agents include, but are not limited to, fatty acids, triglycerides, other like hydrophobic compounds, organic solvents, plasticizing compounds and hydrophilic compounds. Suitable release rate modification agents include, for example, esters of mono-, di-, and tricarboxylic acids, such as 2-ethoxyethyl acetate, methyl acetate, ethyl acetate, diethyl phthalate, dimethyl phthalate, dibutyl phthalate, dimethyl adipate, dimethyl succinate, dimethyl oxalate, dimethyl citrate, triethyl citrate, acetyl tributyl citrate, acetyl triethyl citrate, glycerol triacetate, di(n-butyl) sebecate, and the like; polyhydroxy alcohols, such as propylene glycol, polyethylene glycol, glycerin, sorbitol, and the like; fatty acids; triesters of glycerol, such as triglycerides, epoxidized soybean oil, and other epoxidized vegetable oils; sterols, such as cholesterol; alcohols, such as C.sub.6 -C.sub.12 alkanols, 2-ethoxyethanol. The release rate modification agent may be used singly or in combination with other such agents. Suitable combinations of release rate modification agents include, but are not limited to, glycerin/propylene glycol, sorbitol/glycerine, ethylene oxide/propylene oxide, butylene glycol/adipic acid, and the like. Preferred release rate modification agents include, but are not limited to, dimethyl citrate, triethyl citrate, ethyl heptanoate, glycerin, and hexanediol. Suitable pore-forming agents that may be used in the polymer composition include, but are not limited to, sugars such as sucrose and dextrose, salts such as sodium chloride and sodium carbonate, polymers such as hydroxylpropylcellulose, carboxymethylcellulose, polyethylene glycol, and

polyvinylpyrrolidone. Solid crystals that will provide a defined pore size, such as salt or sugar, are preferred.

In specific preferred embodiments the polypeptide, polynucleotide, and antibody compositions of the invention are formulated using the BEMATM BioErodible Mucoadhesive System, MCATM MucoCutaneous Absorption System, SMPTM Solvent MicroParticle System, or BCPTM BioCompatible Polymer System of Atrix Laboratories, Inc. (Fort Collins, Colorado).

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Sustained-release Therapeutics also include liposomally entrapped Therapeutics of the invention (see generally, Langer, Science 249:1527-1533 (1990); Treat et al., in Liposomes in the Therapy of Infectious Disease and Cancer, Lopez-Berestein and Fidler (eds.), Liss, New York, pp. 317 -327 and 353-365 (1989)). Liposomes containing the Therapeutic are prepared by methods known per se: DE 3,218,121; Epstein et al., Proc. Natl. Acad. Sci. (USA) 82:3688-3692 (1985); Hwang et al., Proc. Natl. Acad. Sci.(USA) 77:4030-4034 (1980); EP 52,322; EP 36,676; EP 88,046; EP 143,949; EP 142,641; Japanese Pat. Appl. 83-118008; U.S. Pat. Nos. 4,485,045 and 4,544,545; and EP 102,324. Ordinarily, the liposomes are of the small (about 200-800 Angstroms) unilamellar type in which the lipid content is greater than about 30 mol. percent cholesterol, the selected proportion being adjusted for the optimal Therapeutic.

In yet an additional embodiment, the Therapeutics of the invention are delivered by way of a pump (see Langer, supra; Sefton, CRC Crit. Ref. Biomed. Eng. 14:201 (1987); Buchwald et al., Surgery 88:507 (1980); Saudek et al., N. Engl. J. Med. 321:574 (1989)).

Other controlled release systems are discussed in the review by Langer (Science 249:1527-1533 (1990)).

For parenteral administration, in one embodiment, the Therapeutic is formulated generally by mixing it at the desired degree of purity, in a unit dosage injectable form (solution, suspension, or emulsion), with a pharmaceutically acceptable carrier, i.e., one that is non-toxic to recipients at the dosages and concentrations employed and is compatible with other ingredients of the formulation. For example, the formulation preferably does not include oxidizing agents and other compounds that are known to be deleterious to the Therapeutic.

Generally, the formulations are prepared by contacting the Therapeutic uniformly and intimately with liquid carriers or finely divided solid carriers or both. Then, if necessary, the product is shaped into the desired formulation. Preferably the carrier is a parenteral carrier, more preferably a solution that is isotonic with the blood of the recipient. Examples of such carrier vehicles include water, saline, Ringer's solution, and dextrose solution. Non-aqueous vehicles such as fixed oils and ethyl oleate are also useful herein, as well as liposomes.

The carrier suitably contains minor amounts of additives such as substances that enhance isotonicity and chemical stability. Such materials are non-toxic to recipients at the dosages and concentrations employed, and include buffers such as phosphate, citrate, succinate, acetic acid, and other organic acids or their salts; antioxidants such as ascorbic acid; low molecular weight (less

than about ten residues) polypeptides, e.g., polyarginine or tripeptides; proteins, such as serum albumin, gelatin, or immunoglobulins; hydrophilic polymers such as polyvinylpyrrolidone; amino acids, such as glycine, glutamic acid, aspartic acid, or arginine; monosaccharides, disaccharides, and other carbohydrates including cellulose or its derivatives, glucose, manose, or dextrins; chelating agents such as EDTA; sugar alcohols such as mannitol or sorbitol; counterions such as sodium; and/or nonionic surfactants such as polysorbates, poloxamers, or PEG.

The Therapeutic is typically formulated in such vehicles at a concentration of about 0.1 mg/ml to 100 mg/ml, preferably 1-10 mg/ml, at a pH of about 3 to 8. It will be understood that the use of certain of the foregoing excipients, carriers, or stabilizers will result in the formation of polypeptide salts.

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Any pharmaceutical used for therapeutic administration can be sterile. Sterility is readily accomplished by filtration through sterile filtration membranes (e.g., 0.2 micron membranes). Therapeutics generally are placed into a container having a sterile access port, for example, an intravenous solution bag or vial having a stopper pierceable by a hypodermic injection needle.

Therapeutics ordinarily will be stored in unit or multi-dose containers, for example, sealed ampoules or vials, as an aqueous solution or as a lyophilized formulation for reconstitution. As an example of a lyophilized formulation, 10-ml vials are filled with 5 ml of sterile-filtered 1% (w/v) aqueous Therapeutic solution, and the resulting mixture is lyophilized. The infusion solution is prepared by reconstituting the lyophilized Therapeutic using bacteriostatic Water-for-Injection.

The invention also provides a pharmaceutical pack or kit comprising one or more containers filled with one or more of the ingredients of the Therapeutics of the invention. Associated with such container(s) can be a notice in the form prescribed by a governmental agency regulating the manufacture, use or sale of pharmaceuticals or biological products, which notice reflects approval by the agency of manufacture, use or sale for human administration. In addition, the Therapeutics may be employed in conjunction with other therapeutic compounds.

The Therapeutics of the invention may be administered alone or in combination with adjuvants. Adjuvants that may be administered with the Therapeutics of the invention include, but are not limited to, alum, alum plus deoxycholate (ImmunoAg), MTP-PE (Biocine Corp.), QS21 (Genentech, Inc.), BCG (e.g., THERACYS®), MPL and nonviable prepartions of Corynebacterium parvum. In a specific embodiment, Therapeutics of the invention are administered in combination with alum. In another specific embodiment, Therapeutics of the invention are administered in combination with QS-21. Further adjuvants that may be administered with the Therapeutics of the invention include, but are not limited to, Monophosphoryl lipid immunomodulator, AdjuVax 100a, QS-21, QS-18, CRL1005, Aluminum salts, MF-59, and Virosomal adjuvant technology. Vaccines that may be administered with the Therapeutics of the invention include, but are not limited to, vaccines directed toward protection against MMR (measles, mumps, rubella), polio, varicella, tetanus/diptheria, hepatitis A, hepatitis

B, haemophilus influenzae B, whooping cough, pneumonia, influenza, Lyme's Disease, rotavirus, cholera, yellow fever, Japanese encephalitis, poliomyelitis, rabies, typhoid fever, and pertussis. Combinations may be administered either concomitantly, e.g., as an admixture, separately but simultaneously or concurrently; or sequentially. This includes presentations in which the combined agents are administered together as a therapeutic mixture, and also procedures in which the combined agents are administered separately but simultaneously, e.g., as through separate intravenous lines into the same individual. Administration "in combination" further includes the separate administration of one of the compounds or agents given first, followed by the second.

The Therapeutics of the invention may be administered alone or in combination with other therapeutic agents. Therapeutic agents that may be administered in combination with the Therapeutics of the invention, include but not limited to, chemotherapeutic agents, antibiotics, steroidal and non-steroidal anti-inflammatories, conventional immunotherapeutic agents, and/or therapeutic treatments described below. Combinations may be administered either concomitantly, e.g., as an admixture, separately but simultaneously or concurrently; or sequentially. This includes presentations in which the combined agents are administered together as a therapeutic mixture, and also procedures in which the combined agents are administered separately but simultaneously, e.g., as through separate intravenous lines into the same individual. Administration "in combination" further includes the separate administration of one of the compounds or agents given first, followed by the second.

In one embodiment, the Therapeutics of the invention are administered in combination with an anticoagulant. Anticoagulants that may be administered with the compositions of the invention include, but are not limited to, heparin, low molecular weight heparin, warfarin sodium (e.g., COUMADIN®), dicumarol, 4-hydroxycoumarin, anisindione (e.g., MIRADONTM), acenocoumarol (e.g., nicoumalone, SINTHROMETM), indan-1,3-dione, phenprocoumon (e.g., MARCUMARTM), ethyl biscoumacetate (e.g., TROMEXANTM), and aspirin. In a specific embodiment, compositions of the invention are administered in combination with heparin and/or warfarin. In another specific embodiment, compositions of the invention are administered in combination with warfarin and aspirin. In another specific embodiment, compositions of the invention are administered in combination with heparin. In another specific embodiment, compositions of the invention are administered in combination with heparin. In another specific embodiment, compositions of the invention are administered in combination with heparin. In another specific embodiment, compositions of the invention are administered in combination with heparin and aspirin.

In another embodiment, the Therapeutics of the invention are administered in combination with thrombolytic drugs. Thrombolytic drugs that may be administered with the compositions of the invention include, but are not limited to, plasminogen, lys-plasminogen, alpha2-antiplasmin, streptokinae (e.g., KABIKINASETM), antiresplace (e.g., EMINASETM), tissue plasminogen activator (t-PA, altevase, ACTIVASETM), urokinase (e.g., ABBOKINASETM), sauruplase,

(Prourokinase, single chain urokinase), and aminocaproic acid (e.g., AMICARTM). In a specific embodiment, compositions of the invention are administered in combination with tissue plasminogen activator and aspirin.

In another embodiment, the Therapeutics of the invention are administered in combination with antiplatelet drugs. Antiplatelet drugs that may be administered with the compositions of the invention include, but are not limited to, aspirin, dipyridamole (e.g., PERSANTINETM), and ticlopidine (e.g., TICLIDTM).

In specific embodiments, the use of anti-coagulants, thrombolytic and/or antiplatelet drugs in combination with Therapeutics of the invention is contemplated for the detection, prevention, diagnosis, prognostication, treatment, and/or amelioration of thrombosis, arterial thrombosis, venous thrombosis, thromboembolism, pulmonary embolism, atherosclerosis, myocardial infarction, transient ischemic attack, unstable angina. In specific embodiments, the use of anticoagulants, thrombolytic drugs and/or antiplatelet drugs in combination with Therapeutics of the invention is contemplated for the prevention of occulsion of saphenous grafts, for reducing the risk of periprocedural thrombosis as might accompany angioplasty procedures, for reducing the risk of stroke in patients with atrial fibrillation including nonrheumatic atrial fibrillation, for reducing the risk of embolism associated with mechanical heart valves and or mitral valves disease. Other uses for the therapeutics of the invention, alone or in combination with antiplatelet, anticoagulant, and/or thrombolytic drugs, include, but are not limited to, the prevention of occlusions in extracorporeal devices (e.g., intravascular canulas, vascular access shunts in hemodialysis patients, hemodialysis machines, and cardiopulmonary bypass machines).

In certain embodiments, Therapeutics of the invention are administered in combination with antiretroviral agents, nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs), non-nucleoside reverse transcriptase inhibitors (NNRTIs), and/or protease inhibitors (PIs). NRTIs that may be administered in combination with the Therapeutics of the invention, include, but are not limited to, RETROVIRTM (zidovudine/AZT), VIDEXTM (didanosine/ddI), HIVIDTM (zalcitabine/ddC), ZERITTM (stavudine/d4T), EPIVIRTM (lamivudine/3TC), and COMBIVIRTM (zidovudine/lamivudine). NNRTIs that may be administered in combination with the Therapeutics of the invention, include, but are not limited to, VIRAMUNETM (nevirapine), RESCRIPTORTM (delavirdine), and SUSTIVATM (efavirenz). Protease inhibitors that may be administered in combination with the Therapeutics of the invention, include, but are not limited to, CRIXIVANTM (indinavir), NORVIRTM (ritonavir), INVIRASETM (saquinavir), and VIRACEPTTM (nelfinavir). In a specific embodiment, antiretroviral agents, nucleoside reverse transcriptase inhibitors, non-nucleoside reverse transcriptase inhibitors, and/or protease inhibitors may be used in any combination with Therapeutics of the invention to treat AIDS and/or to prevent or treat HIV infection.

Additional NRTIs include LODENOSINE™ (F-ddA; an acid-stable adenosine NRTI; Triangle/Abbott; COVIRACIL™ (emtricitabine/FTC; structurally related to lamivudine (3TC) but with 3- to 10-fold greater activity *in vitro*; Triangle/Abbott); dOTC (BCH-10652, also structurally related to lamivudine but retains activity against a substantial proportion of lamivudine-resistant isolates; Biochem Pharma); Adefovir (refused approval for anti-HIV therapy by FDA; Gilead Sciences); PREVEON® (Adefovir Dipivoxil, the active prodrug of adefovir; its active form is PMEA-pp); TENOFOVIR™ (bis-POC PMPA, a PMPA prodrug; Gilead); DAPD/DXG (active metabolite of DAPD; Triangle/Abbott); D-D4FC (related to 3TC, with activity against AZT/3TC-resistant virus); GW420867X (Glaxo Wellcome); ZIAGEN™ (abacavir/159U89; Glaxo Wellcome Inc.); CS-87 (3'azido-2',3'-dideoxyuridine; WO 99/66936); and S-acyl-2-thioethyl (SATE)-bearing prodrug forms of β-L-FD4C and β-L-FddC (WO 98/17281).

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Additional NNRTIs include COACTINON™ (Emivirine/MKC-442, potent NNRTI of the HEPT class; Triangle/Abbott); CAPRAVIRINE™ (AG-1549/S-1153, a next generation NNRTI with activity against viruses containing the K103N mutation; Agouron); PNU-142721 (has 20- to 50-fold greater activity than its predecessor delavirdine and is active against K103N mutants; Pharmacia & Upjohn); DPC-961 and DPC-963 (second-generation derivatives of efavirenz, designed to be active against viruses with the K103N mutation; DuPont); GW-420867X (has 25-fold greater activity than HBY097 and is active against K103N mutants; Glaxo Wellcome); CALANOLIDE A (naturally occurring agent from the latex tree; active against viruses containing either or both the Y181C and K103N mutations); and Propolis (WO 99/49830).

Additional protease inhibitors include LOPINAVIR™ (ABT378/r; Abbott Laboratories); BMS-232632 (an azapeptide; Bristol-Myres Squibb); TIPRANAVIR™ (PNU-140690, a nonpeptic dihydropyrone; Pharmacia & Upjohn); PD-178390 (a nonpeptidic dihydropyrone; Parke-Davis); BMS 232632 (an azapeptide; Bristol-Myers Squibb); L-756,423 (an indinavir analog; Merck); DMP-450 (a cyclic urea compound; Avid & DuPont); AG-1776 (a peptidomimetic with in vitro activity against protease inhibitor-resistant viruses; Agouron); VX-175/GW-433908 (phosphate prodrug of amprenavir; Vertex & Glaxo Welcome); CGP61755 (Ciba); and AGENERASE™ (amprenavir; Glaxo Wellcome Inc.).

Additional antiretroviral agents include fusion inhibitors/gp41 binders. Fusion inhibitors/gp41 binders include T-20 (a peptide from residues 643-678 of the HIV gp41 transmembrane protein ectodomain which binds to gp41 in its resting state and prevents transformation to the fusogenic state; Trimeris) and T-1249 (a second-generation fusion inhibitor; Trimeris).

Additional antiretroviral agents include fusion inhibitors/chemokine receptor antagonists. Fusion inhibitors/chemokine receptor antagonists include CXCR4 antagonists such as AMD 3100 (a bicyclam), SDF-1 and its analogs, and ALX40-4C (a cationic peptide), T22 (an 18 amino acid

peptide; Trimeris) and the T22 analogs T134 and T140; CCR5 antagonists such as RANTES (9-68), AOP-RANTES, NNY-RANTES, and TAK-779; and CCR5/CXCR4 antagonists such as NSC 651016 (a distamycin analog). Also included are CCR2B, CCR3, and CCR6 antagonists. Chemokine recpetor agonists such as RANTES, SDF-1, MIP-1α, MIP-1β, etc., may also inhibit fusion.

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Additional antiretroviral agents include integrase inhibitors. Integrase inhibitors include dicaffeoylquinic (DFQA) acids; L-chicoric acid (a dicaffeoyltartaric (DCTA) acid); quinalizarin (QLC) and related anthraquinones; ZINTEVIRTM (AR 177, an oligonucleotide that probably acts at cell surface rather than being a true integrase inhibitor; Arondex); and naphthols such as those disclosed in WO 98/50347.

Additional antiretroviral agents include hydroxyurea-like compunds such as BCX-34 (a purine nucleoside phosphorylase inhibitor; Biocryst); ribonucleotide reductase inhibitors such as DIDOX™ (Molecules for Health); inosine monophosphate dehydrogenase (IMPDH) inhibitors such as VX-497 (Vertex); and mycopholic acids such as CellCept (mycophenolate mofetil; Roche).

Additional antiretroviral agents include inhibitors of viral integrase, inhibitors of viral genome nuclear translocation such as arylene bis(methylketone) compounds; inhibitors of HIV entry such as AOP-RANTES, NNY-RANTES, RANTES-IgG fusion protein, soluble complexes of RANTES and glycosaminoglycans (GAG), and AMD-3100; nucleocapsid zinc finger inhibitors such as dithiane compounds; targets of HIV Tat and Rev; and pharmacoenhancers such as ABT-378.

Other antiretroviral therapies and adjunct therapies include cytokines and lymphokines such as MIP-1α, MIP-1β, SDF-1α, IL-2, PROLEUKIN™ (aldesleukin/L2-7001; Chiron), IL-4, IL-10, IL-12, and IL-13; interferons such as IFN-α2a; antagonists of TNFs, NFκB, GM-CSF, M-CSF, and IL-10; agents that modulate immune activation such as cyclosporin and prednisone; vaccines such as Remune™ (HIV Immunogen), APL 400-003 (Apollon), recombinant gp120 and fragments, bivalent (B/E) recombinant envelope glycoprotein, rgp120CM235, MN rgp120, SF-2 rgp120, gp120/soluble CD4 complex, Delta JR-FL protein, branched synthetic peptide derived from discontinuous gp120 C3/C4 domain, fusion-competent immunogens, and Gag, Pol, Nef, and Tat vaccines; gene-based therapies such as genetic suppressor elements (GSEs; WO 98/54366), and intrakines (genetically modified CC chemokines targetted to the ER to block surface expression of newly synthesized CCR5 (Yang et al., PNAS 94:11567-72 (1997); Chen et al., Nat. Med. 3:1110-16 (1997)); antibodies such as the anti-CXCR4 antibody 12G5, the anti-CCR5 antibodies 2D7, 5C7, PA8, PA9, PA10, PA11, PA12, and PA14, the anti-CD4 antibodies Q4120 and RPA-T4, the anti-CCR3 antibody 7B11, the anti-gp120 antibodies 17b, 48d, 447-52D, 257-D, 268-D and 50.1, anti-Tat antibodies, anti-TNF-α antibodies, and monoclonal antibody 33A; aryl

PCT/US02/08277 WO 03/038063

hydrocarbon (AH) receptor agonists and antagonists such as TCDD, 3,3',4,4',5pentachlorobiphenyl, 3,3',4,4'-tetrachlorobiphenyl, and α-naphthoflavone (WO 98/30213); and antioxidants such as y-L-glutamyl-L-cysteine ethyl ester (y-GCE; WO 99/56764).

In a further embodiment, the Therapeutics of the invention are administered in combination with an antiviral agent. Antiviral agents that may be administered with the Therapeutics of the invention include, but are not limited to, acyclovir, ribavirin, amantadine, and

remantidine.

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In other embodiments, Therapeutics of the invention may be administered in combination with anti-opportunistic infection agents. Anti-opportunistic agents that may be administered in combination with the Therapeutics of the invention, include, but are not limited to, DAPSONE™, PENTAMIDINE™, TRIMETHOPRIM-SULFAMETHOXAZOLE™, ATOVAQUONE™, ISONIAZID™, RIFAMPIN™, PYRAZINAMIDE™, ETHAMBUTOL™, CLARITHROMYCIN™, AZITHROMYCIN™, RIFABUTIN™. GANCICLOVIR™, ITRACONAZOLE™, FLUCONAZOLE™, FOSCARNET™, CIDOFOVIR™, PYRIMETHAMINE™, KETOCONAZOLE™, ACYCLOVIR™, FAMCICOLVIR™, LEUCOVORIN™, NEUPOGEN™ (filgrastim/G-CSF), and LEUKINE™ (sargramostim/GM-CSF). In a specific embodiment, Therapeutics of the invention are used in any combination with TRIMETHOPRIM-SULFAMETHOXAZOLE™, DAPSONE™, PENTAMIDINE™, and/or ATOVAOUONE™ to prophylactically treat or prevent an opportunistic Pneumocystis carinii pneumonia infection. In another specific embodiment, Therapeutics of the invention are used in with ISONIAZID™, RIFAMPIN™, PYRAZINAMIDE™, and/or combination ETHAMBUTOL™ to prophylactically treat or prevent an opportunistic Mycobacterium avium complex infection. In another specific embodiment, Therapeutics of the invention are used in any combination with RIFABUTIN™, CLARITHROMYCIN™, and/or AZITHROMYCIN™ to prophylactically treat or prevent an opportunistic Mycobacterium tuberculosis infection. In another specific embodiment, Therapeutics of the invention are used in any combination with GANCICLOVIR™, FOSCARNET™, and/or CIDOFOVIR™ to prophylactically treat or prevent an opportunistic cytomegalovirus infection. In another specific embodiment, Therapeutics of the invention are used in any combination with FLUCONAZOLE™, ITRACONAZOLE™, and/or KETOCONAZOLE™ to prophylactically treat or prevent an opportunistic fungal infection. In another specific embodiment, Therapeutics of the invention are used in any combination with ACYCLOVIR™ and/or FAMCICOLVIR™ to prophylactically treat or prevent an opportunistic herpes simplex virus type I and/or type II infection. In another specific embodiment, Therapeutics of the invention are used in any combination with PYRIMETHAMINE™ and/or LEUCOVORIN™ to prophylactically treat or prevent an opportunistic Toxoplasma gondii infection. In another

specific embodiment, Therapeutics of the invention are used in any combination with LEUCOVORIN™ and/or NEUPOGEN™ to prophylactically treat or prevent an opportunistic bacterial infection.

In a further embodiment, the Therapeutics of the invention are administered in combination with an antibiotic agent. Antibiotic agents that may be administered with the Therapeutics of the invention include, but are not limited to, amoxicillin, beta-lactamases, aminoglycosides, beta-lactam (glycopeptide), beta-lactamases, Clindamycin, chloramphenicol, cephalosporins, ciprofloxacin, erythromycin, fluoroquinolones, macrolides, metronidazole, penicillins, quinolones, rapamycin, rifampin, streptomycin, sulfonamide, tetracyclines, trimethoprim, trimethoprim-sulfamethoxazole, and vancomycin.

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In other embodiments, the Therapeutics of the invention are administered in combination with immunestimulants. Immunostimulants that may be administered in combination with the Therapeutics of the invention include, but are not limited to, levamisole (e.g., ERGAMISOLTM), isoprinosine (e.g. INOSIPLEXTM), interferons (e.g. interferon alpha), and interleukins (e.g., IL-2).

In other embodiments, Therapeutics of the invention are administered in combination with immunosuppressive agents. Immunosuppressive agents that may be administered in combination with the Therapeutics of the invention include, but are not limited to, steroids, cyclosporine, cyclosporine analogs, cyclophosphamide methylprednisone, prednisone, azathioprine, FK-506, 15deoxyspergualin, and other immunosuppressive agents that act by suppressing the function of responding T cells. Other immunosuppressive agents that may be administered in combination with the Therapeutics of the invention include, but are not limited to, prednisolone, methotrexate, thalidomide, methoxsalen, rapamycin, leflunomide, mizoribine (BREDININTM), brequinar, deoxyspergualin, and azaspirane (SKF 105685), ORTHOCLONE OKT® 3 (muromonab-CD3), SANDIMMUNE™, NEORAL™, SANGDYA™ (cyclosporine), PROGRAF® (FK506, tacrolimus), CELLCEPT® (mycophenolate motefil, of which the active metabolite is mycophenolic acid), IMURANTM (azathioprine), glucocorticosteroids, adrenocortical steroids such as DELTASONE™ (prednisone) and HYDELTRASOL™ (prednisolone), FOLEX™ and MEXATE™ (methotrxate), OXSORALEN-ULTRA™ (methoxsalen) and RAPAMUNE™ (sirolimus). In a specific embodiment, immunosuppressants may be used to prevent rejection of organ or bone marrow transplantation.

In an additional embodiment, Therapeutics of the invention are administered alone or in combination with one or more intravenous immune globulin preparations. Intravenous immune globulin preparations that may be administered with the Therapeutics of the invention include, but not limited to, GAMMARTM, IVEEGAMTM, SANDOGLOBULINTM, GAMMAGARD S/DTM, ATGAMTM (antithymocyte glubulin), and GAMIMUNETM. In a specific embodiment,

Therapeutics of the invention are administered in combination with intravenous immune globulin preparations in transplantation therapy (e.g., bone marrow transplant).

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In certain embodiments, the Therapeutics of the invention are administered alone or in combination with an anti-inflammatory agent. Anti-inflammatory agents that may be administered with the Therapeutics of the invention include, but are not limited to, corticosteroids (e.g. betamethasone, budesonide, cortisone, dexamethasone, hydrocortisone, methylprednisolone, prednisolone, prednisone, and triamcinolone), nonsteroidal anti-inflammatory drugs (e.g., diclofenac, diflunisal, etodolac, fenoprofen, floctafenine, flurbiprofen, ibuprofen, indomethacin, ketoprofen, meclofenamate, mefenamic acid, meloxicam, nabumetone, naproxen, oxaprozin, phenylbutazone, piroxicam, sulindac, tenoxicam, tiaprofenic acid, and tolmetin.), as well as antihistamines, aminoarylcarboxylic acid derivatives, arylacetic acid derivatives, arylbutyric acid derivatives, arylcarboxylic acids, arylpropionic acid derivatives, pyrazoles, pyrazolones, salicylic acid derivatives, thiazinecarboxamides, e-acetamidocaproic acid, S-adenosylmethionine, 3-amino-4-hydroxybutyric acid, amixetrine, bendazac, benzydamine, bucolome, difenpiramide, ditazol, emorfazone, guaiazulene, nabumetone, nimesulide, orgotein, oxaceprol, paranyline, perisoxal, pifoxime, proquazone, proxazole, and tenidap.

In an additional embodiment, the compositions of the invention are administered alone or in combination with an anti-angiogenic agent. Anti-angiogenic agents that may be administered with the compositions of the invention include, but are not limited to, Angiostatin (Entremed, Rockville, MD), Troponin-1 (Boston Life Sciences, Boston, MA), anti-Invasive Factor, retinoic acid and derivatives thereof, paclitaxel (Taxol), Suramin, Tissue Inhibitor of Metalloproteinase-1, Tissue Inhibitor of Metalloproteinase-2, VEGI, Plasminogen Activator Inhibitor-1, Plasminogen Activator Inhibitor-2, and various forms of the lighter "d group" transition metals.

Lighter "d group" transition metals include, for example, vanadium, molybdenum, tungsten, titanium, niobium, and tantalum species. Such transition metal species may form transition metal complexes. Suitable complexes of the above-mentioned transition metal species include oxo transition metal complexes.

Representative examples of vanadium complexes include oxo vanadium complexes such as vanadate and vanadyl complexes. Suitable vanadate complexes include metavanadate and orthovanadate complexes such as, for example, ammonium metavanadate, sodium metavanadate, and sodium orthovanadate. Suitable vanadyl complexes include, for example, vanadyl acetylacetonate and vanadyl sulfate including vanadyl sulfate hydrates such as vanadyl sulfate mono- and trihydrates.

Representative examples of tungsten and molybdenum complexes also include oxo complexes. Suitable oxo tungsten complexes include tungstate and tungsten oxide complexes. Suitable tungstate complexes include ammonium tungstate, calcium tungstate, sodium tungstate dihydrate, and tungstic acid. Suitable tungsten oxides include tungsten (IV) oxide and tungsten

(VI) oxide. Suitable oxo molybdenum complexes include molybdate, molybdenum oxide, and molybdenyl complexes. Suitable molybdate complexes include ammonium molybdate and its hydrates, sodium molybdate and its hydrates, and potassium molybdate and its hydrates. Suitable molybdenum oxides include molybdenum (VI) oxide, molybdenum (VI) oxide, and molybdic acid. Suitable molybdenyl complexes include, for example, molybdenyl acetylacetonate. Other suitable tungsten and molybdenum complexes include hydroxo derivatives derived from, for example, glycerol, tartaric acid, and sugars.

A wide variety of other anti-angiogenic factors may also be utilized within the context of the present invention. Representative examples include, but are not limited to, platelet factor 4; protamine sulphate; sulphated chitin derivatives (prepared from queen crab shells), (Murata et al., Cancer Res. 51:22-26, (1991)); Sulphated Polysaccharide Peptidoglycan Complex (SP-PG) (the function of this compound may be enhanced by the presence of steroids such as estrogen, and tamoxifen citrate); Staurosporine; modulators of matrix metabolism, including for example, proline analogs, cishydroxyproline, d,L-3,4-dehydroproline, Thiaproline, alpha,alpha-dipyridyl, 4-propyl-5-(4-pyridinyl)-2(3H)-oxazolone; aminopropionitrile fumarate; Mitoxantrone; Heparin; Interferons; 2 Macroglobulin-serum; ChIMP-3 (Pavloff et al., J. Bio. Chem. 267:17321-17326, (1992)); Chymostatin (Tomkinson et al., Biochem J. 286:475-480, (1992)); Cyclodextrin Tetradecasulfate; Eponemycin; Camptothecin; Fumagillin (Ingber et al., Nature 348:555-557, (1990)); Gold Sodium Thiomalate ("GST"; Matsubara and Ziff, J. Clin. Invest. 79:1440-1446, (1987)); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664, (1987)); Bisantrene (National Cancer Institute); Lobenzarit disodium (N-(2)-carboxyphenyl-4- chloroanthronilic acid disodium or "CCA"; (Takeuchi et al., Agents Actions 36:312-316, (1992)); and metalloproteinase inhibitors such as BB94.

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Additional anti-angiogenic factors that may also be utilized within the context of the present invention include Thalidomide, (Celgene, Warren, NJ); Angiostatic steroid; AGM-1470 (H. Brem and J. Folkman J Pediatr. Surg. 28:445-51 (1993)); an integrin alpha v beta 3 antagonist (C. Storgard et al., J Clin. Invest. 103:47-54 (1999)); carboxynaminolmidazole; Carboxyamidotriazole (CAI) (National Cancer Institute, Bethesda, MD); Conbretastatin A-4 (CA4P) (OXiGENE, Boston, MA); Squalarnine (Magainin Pharmaceuticals, Plymouth Meeting, PA); TNP-470, (Tap Pharmaceuticals, Deerfield, IL); ZD-0101 AstraZeneca (London, UK); APRA (CT2584); Benefin, Byrostatin-1 (SC339555); CGP-41251 (PKC 412); CM101; Dexrazoxane (ICRF187); DMXAA; Endostatin; Flavopridiol; Genestein; GTE; ImmTher; Iressa (ZD1839); Octreotide (Somatostatin); Panretin; Penacillamine; Photopoint; PI-88; Prinomastat (AG-3340) Purlytin; Suradista (FCE26644); Tamoxifen (Nolvadex); Tazarotene; Tetrathiomolybdate; Xeloda (Capecitabine); and 5-Fluorouracil.

Anti-angiogenic agents that may be administed in combination with the compounds of the invention may work through a variety of mechanisms including, but not limited to, inhibiting

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proteolysis of the extracellular matrix, blocking the function of endothelial cell-extracellular matrix adhesion molecules, by antagonizing the function of angiogenesis inducers such as growth factors, and inhibiting integrin receptors expressed on proliferating endothelial cells. Examples of anti-angiogenic inhibitors that interfere with extracellular matrix proteolysis and which may be administered in combination with the compositons of the invention include, but are not lmited to, AG-3340 (Agouron, La Jolla, CA), BAY-12-9566 (Bayer, West Haven, CT), BMS-275291 (Bristol Myers Squibb, Princeton, NJ), CGS-27032A (Novartis, East Hanover, NJ), Marimastat (British Bjotech, Oxford, UK), and Metastat (Aeterna, St-Foy, Quebec). Examples of antiangiogenic inhibitors that act by blocking the function of endothelial cell-extracellular matrix adhesion molecules and which may be administered in combination with the compositons of the invention include, but are not lmited to, EMD-121974 (Merck KcgaA Darmstadt, Germany) and Vitaxin (Ixsys, La Jolla, CA/Medimmune, Gaithersburg, MD). Examples of anti-angiogenic agents that act by directly antagonizing or inhibiting angiogenesis inducers and which may be administered in combination with the compositons of the invention include, but are not lmited to, Angiozyme (Ribozyme, Boulder, CO), Anti-VEGF antibody (Genentech, S. San Francisco, CA), PTK-787/ZK-225846 (Novartis, Basel, Switzerland), SU-101 (Sugen, S. San Francisco, CA), SU-5416 (Sugen/ Pharmacia Upjohn, Bridgewater, NJ), and SU-6668 (Sugen). Other anti-angiogenic agents act to indirectly inhibit angiogenesis. Examples of indirect inhibitors of angiogenesis which may be administered in combination with the compositons of the invention include, but are not limited to, IM-862 (Cytran, Kirkland, WA), Interferon-alpha, IL-12 (Roche, Nutley, NJ), and Pentosan polysulfate (Georgetown University, Washington, DC).

In particular embodiments, the use of compositions of the invention in combination with anti-angiogenic agents is contemplated for the treatment, prevention, and/or amelioration of an autoimmune disease, such as for example, an autoimmune disease described herein.

In a particular embodiment, the use of compositions of the invention in combination with anti-angiogenic agents is contemplated for the treatment, prevention, and/or amelioration of arthritis. In a more particular embodiment, the use of compositions of the invention in combination with anti-angiogenic agents is contemplated for the treatment, prevention, and/or amelioration of rheumatoid arthritis.

In another embodiment, the polynucleotides encoding a polypeptide of the present invention are administered in combination with an angiogenic protein, or polynucleotides encoding an angiogenic protein. Examples of angiogenic proteins that may be administered with the compositions of the invention include, but are not limited to, acidic and basic fibroblast growth factors, VEGF-1, VEGF-2, VEGF-3, epidermal growth factor alpha and beta, platelet-derived endothelial cell growth factor, platelet-derived growth factor, tumor necrosis factor alpha, hepatocyte growth factor, insulin-like growth factor, colony stimulating factor, macrophage colony stimulating factor, granulocyte/macrophage colony stimulating factor, and nitric oxide synthase.

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In additional embodiments, compositions of the invention are administered in combination with a chemotherapeutic agent. Chemotherapeutic agents that may be administered with the Therapeutics of the invention include, but are not limited to alkylating agents such as nitrogen mustards (for example, Mechlorethamine, cyclophosphamide, Cyclophosphamide Ifosfamide, Melphalan (L-sarcolysin), and Chlorambucil), ethylenimines and methylmelamines (for example, Hexamethylmelamine and Thiotepa), alkyl sulfonates (for example, Busulfan), nitrosoureas (for example, Carmustine (BCNU), Lomustine (CCNU), Semustine (methyl-CCNU), and Streptozocin Dacarbazine (DTIC; (streptozotocin)), triazenes . (for example, dimethyltriazenoimidazolecarboxamide)), folic acid analogs (for example, Methotrexate (amethopterin)), pyrimidine analogs (for example, Fluorouacil (5-fluorouracil; 5-FU), Floxuridine (fluorodeoxyuridine; FudR), and Cytarabine (cytosine arabinoside)), purine analogs and related inhibitors (for example, Mercaptopurine (6-mercaptopurine; 6-MP), Thioguanine (6-thioguanine; TG), and Pentostatin (2'-deoxycoformycin)), vinca alkaloids (for example, Vinblastine (VLB, vinblastine sulfate)) and Vincristine (vincristine sulfate)), epipodophyllotoxins (for example, Etoposide and Teniposide), antibiotics (for example, Dactinomycin (actinomycin D), Daunorubicin (daunomycin; rubidomycin), Doxorubicin, Bleomycin, Plicamycin (mithramycin), and Mitomycin (mitomycin C), enzymes (for example, L-Asparaginase), biological response modifiers (for example, Interferon-alpha and interferon-alpha-2b), platinum coordination compounds (for example, Cisplatin (cis-DDP) and Carboplatin), anthracenedione (Mitoxantrone), substituted ureas (for example, Hydroxyurea), methylhydrazine derivatives (for example, Procarbazine (N-methylhydrazine; MIH), adrenocorticosteroids (for example, Prednisone), progestins (for example, Hydroxyprogesterone caproate, Medroxyprogesterone, Medroxyprogesterone acetate, and Megestrol acetate), estrogens (for example, Diethylstilbestrol (DES), Diethylstilbestrol diphosphate, Estradiol, and Ethinyl estradiol), antiestrogens (for example, Tamoxifen), androgens (Testosterone proprionate, and Fluoxymesterone), antiandrogens (for example, Flutamide), gonadotropin-releasing horomone analogs (for example, Leuprolide), other hormones and hormone analogs (for example, methyltestosterone, estramustine, estramustine phosphate sodium, chlorotrianisene, and testolactone), and others (for example, dicarbazine, glutamic acid, and mitotane).

In one embodiment, the compositions of the invention are administered in combination with one or more of the following drugs: infliximab (also known as RemicadeTM Centocor, Inc.), Trocade (Roche, RO-32-3555), Leflunomide (also known as AravaTM from Hoechst Marion Roussel), KineretTM (an IL-1 Receptor antagonist also known as Anakinra from Amgen, Inc.)

In a specific embodiment, compositions of the invention are administered in combination with CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisone) or combination of one or more of the components of CHOP. In one embodiment, the compositions of the invention are administered in combination with anti-CD20 antibodies, human monoclonal anti-CD20 antibodies.

In another embodiment, the compositions of the invention are administered in combination with anti-CD20 antibodies and CHOP, or anti-CD20 antibodies and any combination of one or more of the components of CHOP, particularly cyclophosphamide and/or prednisone. In a specific embodiment, compositions of the invention are administered in combination with Rituximab. In a further embodiment, compositions of the invention are administered with Rituximab and CHOP, or Rituximab and any combination of one or more of the components of CHOP, particularly cyclophosphamide and/or prednisone. In a specific embodiment, compositions of the invention are administered in combination with tositumomab. In a further embodiment, compositions of the invention are administered with tositumomab and CHOP, or tositumomab and any combination of one or more of the components of CHOP, particularly cyclophosphamide and/or prednisone. The anti-CD20 antibodies may optionally be associated with radioisotopes, toxins or cytotoxic prodrugs.

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In another specific embodiment, the compositions of the invention are administered in combination ZevalinTM. In a further embodiment, compositions of the invention are administered with ZevalinTM and CHOP, or ZevalinTM and any combination of one or more of the components of CHOP, particularly cyclophosphamide and/or prednisone. ZevalinTM may be associated with one or more radisotopes. Particularly preferred isotopes are ⁹⁰Y and ¹¹¹In.

In an additional embodiment, the Therapeutics of the invention are administered in combination with cytokines. Cytokines that may be administered with the Therapeutics of the invention include, but are not limited to, IL2, IL3, IL4, IL5, IL6, IL7, IL10, IL12, IL13, IL15, anti-CD40, CD40L, IFN-gamma and TNF-alpha. In another embodiment, Therapeutics of the invention may be administered with any interleukin, including, but not limited to, IL-1alpha, IL-1beta, IL-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-8, IL-9, IL-10, IL-11, IL-12, IL-13, IL-14, IL-15, IL-16, IL-17, IL-18, IL-19, IL-20, and IL-21.

In one embodiment, the Therapeutics of the invention are administered in combination with members of the TNF family. TNF, TNF-related or TNF-like molecules that may be administered with the Therapeutics of the invention include, but are not limited to, soluble forms of TNF-alpha, lymphotoxin-alpha (LT-alpha, also known as TNF-beta), LT-beta (found in complex heterotrimer LT-alpha2-beta), OPGL, FasL, CD27L, CD30L, CD40L, 4-1BBL, DcR3, OX40L, TNF-gamma (International Publication No. WO 96/14328), AIM-I (International Publication No. WO 97/33899), endokine-alpha (International Publication No. WO 98/07880), OPG, and neutrokine-alpha (International Publication No. WO 98/18921, OX40, and nerve growth factor (NGF), and soluble forms of Fas, CD30, CD27, CD40 and 4-IBB, TR2 (International Publication No. WO 96/34095), DR3 (International Publication No. WO 97/33904), DR4 (International Publication No. WO 98/32856), TR5 (International Publication No. WO 98/30693), TRANK, TR9 (International Publication No. WO 98/56892),TR10 (International Publication No. No. WO 98/30693)

WO 98/54202), 312C2 (International Publication No. WO 98/06842), and TR12, and soluble forms CD154, CD70, and CD153.

In an additional embodiment, the Therapeutics of the invention are administered in combination with angiogenic proteins. Angiogenic proteins that may be administered with the Therapeutics of the invention include, but are not limited to, Glioma Derived Growth Factor (GDGF), as disclosed in European Patent Number EP-399816; Platelet Derived Growth Factor-A (PDGF-A), as disclosed in European Patent Number EP-682110; Platelet Derived Growth Factor-B (PDGF-B), as disclosed in European Patent Number EP-282317; Placental Growth Factor (PIGF), as disclosed in International Publication Number WO 92/06194; Placental Growth Factor-2 (PIGF-2), as disclosed in Hauser et al., Growth Factors, 4:259-268 (1993); Vascular Endothelial Growth Factor (VEGF), as disclosed in International Publication Number WO 90/13649; Vascular Endothelial Growth Factor-A (VEGF-A), as disclosed in European Patent Number EP-506477; Vascular Endothelial Growth Factor-2 (VEGF-2), as disclosed in International Publication Number WO 96/39515; Vascular Endothelial Growth Factor B (VEGF-3); Vascular Endothelial Growth Factor B-186 (VEGF-B186), as disclosed in International Publication Number WO 96/26736; Vascular Endothelial Growth Factor-D (VEGF-D), as disclosed in International Publication Number WO 98/02543; Vascular Endothelial Growth Factor-D (VEGF-D), as disclosed in International Publication Number WO 98/07832; and Vascular Endothelial Growth Factor-E (VEGF-E), as disclosed in German Patent Number DE19639601. The above mentioned references are herein incorporated by reference in their entireties.

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In an additional embodiment, the Therapeutics of the invention are administered in combination with Fibroblast Growth Factors. Fibroblast Growth Factors that may be administered with the Therapeutics of the invention include, but are not limited to, FGF-1, FGF-2, FGF-3, FGF-4, FGF-5, FGF-6, FGF-7, FGF-8, FGF-9, FGF-10, FGF-11, FGF-12, FGF-13, FGF-14, and FGF-15.

In an additional embodiment, the Therapeutics of the invention are administered in combination with hematopoietic growth factors. Hematopoietic growth factors that may be administered with the Therapeutics of the invention include, but are not limited to, granulocyte macrophage colony stimulating factor (GM-CSF) (sargramostim, LEUKINETM, PROKINETM), granulocyte colony stimulating factor (G-CSF) (filgrastim, NEUPOGENTM), macrophage colony stimulating factor (M-CSF, CSF-1) erythropoietin (epoetin alfa, EPOGENTM, PROCRITTM), stem cell factor (SCF, c-kit ligand, steel factor), megakaryocyte colony stimulating factor, PIXY321 (a GMCSF/IL-3 fusion protein), interleukins, especially any one or more of IL-1 through IL-12, interferon-gamma, or thrombopoietin.

In certain embodiments, Therapeutics of the present invention are administered in combination with adrenergic blockers, such as, for example, acebutolol, atenolol, betaxolol,

bisoprolol, carteolol, labetalol, metoprolol, nadolol, oxprenolol, penbutolol, pindolol, propranolol, sotalol, and timolol.

In another embodiment, the Therapeutics of the invention are administered in combination with an antiarrhythmic drug (e.g., adenosine, amidoarone, bretylium, digitalis, digoxin, digitoxin, diliazem, disopyramide, esmolol, flecainide, lidocaine, mexiletine, moricizine, phenytoin, procainamide, N-acetyl procainamide, propafenone, propranolol, quinidine, sotalol, tocainide, and verapamil).

In another embodiment, the Therapeutics of the invention are administered in combination with diuretic agents, such as carbonic anhydrase-inhibiting agents (e.g., acetazolamide, dichlorphenamide, and methazolamide), osmotic diuretics (e.g., glycerin, isosorbide, mannitol, and urea), diuretics that inhibit Na*-K*-2Cl symport (e.g., furosemide, bumetanide, azosemide, piretanide, tripamide, ethacrynic acid, muzolimine, and torsemide), thiazide and thiazide-like diuretics (e.g., bendroflumethiazide, benzthiazide, chlorothiazide, hydrochlorothiazide, hydroflumethiazide, methyclothiazide, polythiazide, trichormethiazide, chlorothalidone, indapamide, metolazone, and quinethazone), potassium sparing diuretics (e.g., amiloride and triamterene), and mineralcorticoid receptor antagonists (e.g., spironolactone, canrenone, and potassium canrenoate).

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In one embodiment, the Therapeutics of the invention are administered in combination with treatments for endocrine and/or hormone imbalance disorders. Treatments for endocrine and/or hormone imbalance disorders include, but are not limited to, 127 I, radioactive isotopes of iodine such as 131 and 123 I; recombinant growth hormone, such as HUMATROPE™ (recombinant somatropin); growth hormone analogs such as PROTROPIN™ (somatrem); dopamine agonists such as PARLODEL™ (bromocriptine); somatostatin analogs such as SANDOSTATIN™ (octreotide); gonadotropin preparations such as PREGNYL™, A.P.L.™ and PROFASI™ (chorionic gonadotropin (CG)), PERGONAL™ (menotropins), and METRODIN™ (urofollitropin (uFSH)); synthetic human gonadotropin releasing hormone preparations such as FACTREL™ and LUTREPULSE™ (gonadorelin hydrochloride); synthetic gonadotropin agonists such as LUPRON™ (leuprolide acetate), SUPPRELIN™ (histrelin acetate), SYNAREL™ (nafarelin acetate), and ZOLADEX™ (goserelin acetate); synthetic preparations of thyrotropin-releasing hormone such as RELEFACT TRH™ and THYPINONE™ (protirelin); recombinant human TSH such as THYROGEN™; synthetic preparations of the sodium salts of the natural isomers of thyroid hormones such as L-T₄™, SYNTHROID™ and LEVOTHROID™ (levothyroxine sodium), L-T₃™, CYTOMEL™ and TRIOSTAT™ (liothyroine sodium), and THYROLAR™ (liotrix); antithyroid compounds such as 6-n-propylthiouracil (propylthiouracil), 1-methyl-2mercaptoimidazole and TAPAZOLE™ (methimazole), NEO-MERCAZOLE™ (carbimazole); beta-adrenergic receptor antagonists such as propranolol and esmolol; Ca2+ channel blockers;

dexamethasone and iodinated radiological contrast agents such as TELEPAQUE™ (iopanoic acid) and ORAGRAFIN™ (sodium ipodate).

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Additional treatments for endocrine and/or hormone imbalance disorders include, but are not limited to, estrogens or congugated estrogens such as ESTRACE™ (estradiol), ESTINYL™ (ethinyl estradiol), PREMARIN™, ESTRATAB™, ORTHO-EST™, OGEN™ and estropipate (estrone), ESTROVIS™ (quinestrol), ESTRADERM™ (estradiol), DELESTROGEN™ and VALERGEN™ (estradiol valerate), DEPO-ESTRADIOL CYPIONATE™ and ESTROJECT LA™ (estradiol cypionate); antiestrogens such as NOLVADEX™ (tamoxifen), SEROPHENE™ and CLOMID™ (clomiphene); progestins such as DURALUTIN™ (hydroxyprogesterone caproate), MPA™ and DEPO-PROVERA™ (medroxyprogesterone acetate), PROVERA™ and CYCRIN™ (MPA), MEGACE™ (megestrol acetate), NORLUTIN™ (norethindrone), and NORLUTATE™ and AYGESTIN™ (norethindrone acetate); progesterone implants such as NORPLANT SYSTEM™ (subdermal implants of norgestrel); antiprogestins such as RU 486™ (mifepristone); hormonal contraceptives such as ENOVID™ (norethynodrel plus mestranol), PROGESTASERT™ (intrauterine device that releases progesterone), LOESTRIN™, BREVICON™, MODICON™, GENORA™, NELONA™, NORINYL™, OVACON-35™ and OVACON-50™ (ethinyl estradiol/norethindrone), LEVLEN™, NORDETTE™, TRI-LEVLEN™ and TRIPHASIL-21™ (ethinyl estradiol/levonorgestrel) LO/OVRAL™ and OVRAL™ (ethinyl estradiol/norgestrel), DEMULEN™ (ethinyl estradiol/ethynodiol diacetate), NORINYL™, ORTHO-NOVUM™, NORETHIN™, GENORA™, and NELOVA™ (norethindrone/mestranol), DESOGEN™ and ORTHO-CYCLEN™ ORTHO-CEPT™ (ethinyl estradiol/desogestrel), TRICYCLEN™ (ethinyl estradiol/norgestimate), MICRONOR™ and NOR-QD™ (norethindrone), and OVRETTE™ (norgestrel).

Additional treatments for endocrine and/or hormone imbalance disorders include, but are not limited to, testosterone esters such as methenolone acetate and testosterone undecanoate; parenteral and oral androgens such as TESTOJECT-50TM (testosterone), TESTEXTM (testosterone propionate), DELATESTRYLTM (testosterone enanthate), DEPO-TESTOSTERONETM (testosterone cypionate), DANOCRINETM (danazol), HALOTESTINTM (fluoxymesterone), ORETON METHYLTM, TESTREDTM and VIRILONTM (methyltestosterone), and OXANDRINTM (oxandrolone); testosterone transdermal systems such as TESTODERMTM; androgen receptor antagonist and 5-alpha-reductase inhibitors such as ANDROCURTM (cyproterone acetate), EULEXINTM (flutamide), and PROSCARTM (finasteride); adrenocorticotropic hormone preparations such as CORTROSYNTM (cosyntropin); adrenocortical steroids and their synthetic analogs such as ACLOVATETM (alclometasone dipropionate), CYCLOCORTTM (amcinonide), BECLOVENTTM and VANCERILTM (beclomethasone dipropionate), CELESTONETM

(betamethasone), BENISONE™ and UTICORT™ (betamethasone benzoate), DIPROSONE™ (betamethasone dipropionate), CELESTONE PHOSPHATE™ (betamethasone sodium phosphate), CELESTONE SOLUSPAN™ (betamethasone sodium phosphate and acetate), BETA-VAL™ and VALISONE™ (betamethasone valerate), TEMOVATE™ (clobetasol propionate), CLODERM™ (clocortolone pivalate), CORTEF™ and HYDROCORTONE™ (cortisol (hydrocortisone)), 5 HYDROCORTONE ACETATE™ (cortisol (hydrocortisone) acetate), LOCOID™ (cortisol (hydrocortisone) butyrate), HYDROCORTONE PHOSPHATE™ (cortisol (hydrocortisone) sodium phosphate), A-HYDROCORT™ and SOLU CORTEF™ (cortisol (hydrocortisone) sodium succinate). WESTCORT™ (cortisol (hydrocortisone) valerate), CORTISONE ACETATE™ acetate), DESOWEN™ and TRIDESILON™ (desonide), TOPICORT™ 10 (cortisone (desoximetasone), DECADRON™ (dexamethasone), DECADRON LA™ (dexamethasone acetate), DECADRON PHOSPHATE™ and HEXADROL PHOSPHATE™ (dexamethasone sodium phosphate), FLORONE™ and MAXIFLOR™ (diflorasone diacetate), FLORINEF ACETATE™ (fludrocortisone acetate), AEROBID™ and NASALIDE™ (flunisolide), FLUONID™ and SYNALAR™ (fluocinolone acetonide), LIDEX™ (fluocinonide), FLUOR-OP™ 15 and FML™ (fluorometholone), CORDRAN™ (flurandrenolide), HALOG™ (halcinonide), HMS LIZUIFILM™ (medrysone), MEDROL™ (methylprednisolone), DEPO-MEDROL™ and MEDROL ACETATE™ (methylprednisone acetate), A-METHAPRED™ and SOLUMEDROL™ (methylprednisolone sodium succinate), ELOCON™ (mometasone furoate), HALDRONE™ (paramethasone acetate), DELTA-CORTEF™ (prednisolone), ECONOPRED™ (prednisolone 20 acetate), HYDELTRASOL™ (prednisolone sodium phosphate), HYDELTRA-T.B.A™ (prednisolone tebutate), DELTASONE™ (prednisone), ARISTOCORT™ and KENACORT™ (triamcinolone), KENALOG™ (triamcinolone acetonide), ARISTOCORT™ and KENACORT DIACETATE™ (triamcinolone diacetate), and ARISTOSPAN™ (triamcinolone hexacetonide); inhibitors of biosynthesis and action of adrenocortical steroids such as CYTADREN™ 25 (aminoglutethimide), NIZORAL™ (ketoconazole), MODRASTANE™ (trilostane), METOPIRONE™ (metyrapone); bovine, porcine or human insulin or mixtures thereof; insulin analogs; recombinant human insulin such as HUMULIN™ and NOVOLIN™; oral hypoglycemic agents such as ORAMIDE™ and ORINASE™ (tolbutamide), DIABINESE™ (chlorpropamide), TOLAMIDE™ and TOLINASE™ (tolazamide), DYMELOR™ (acetohexamide), glibenclamide, 30 MICRONASE™, DIBETA™ and GLYNASE™ (glyburide), GLUCOTROL™ (glipizide), and DIAMICRON™ (gliclazide), GLUCOPHAGE™ (metformin), ciglitazone, pioglitazone, and alpha-glucosidase inhibitors; bovine or porcine glucagon; somatostatins such as SANDOSTATIN™ (octreotide); and diazoxides such as PROGLYCEM™ (diazoxide).

In an additional embodiment, the Therapeutics of the invention are administered in combination with drugs effective in treating iron deficiency and hypochromic anemias, including but not limited to, ferrous sulfate (iron sulfate, FEOSOLTM), ferrous fumarate (e.g., FEOSTATTM), ferrous gluconate (e.g., FERGONTM), polysaccharide-iron complex (e.g., NIFEREXTM), iron dextran injection (e.g., INFEDTM), cupric sulfate, pyroxidine, riboflavin, Vitamin B₁₂, cyancobalamin injection (e.g., REDISOLTM, RUBRAMIN PCTM), hydroxocobalamin, folic acid (e.g., FOLVITETM), leucovorin (folinic acid, 5-CHOH4PteGlu, citrovorum factor) or WELLCOVORIN (Calcium salt of leucovorin), transferrin or ferritin.

In another embodiment, Therapeutics of the invention are administered in combination with vasodilating agents and/or calcium channel blocking agents. Vasodilating agents that may be administered with the Therapeutics of the invention include, but are not limited to, Angiotensin Converting Enzyme (ACE) inhibitors (e.g., papaverine, isoxsuprine, benazepril, captopril, cilazapril, enalapril, enalaprilat, fosinopril, lisinopril, moexipril, perindopril, quinapril, ramipril, spirapril, trandolapril, and nylidrin), and nitrates (e.g., isosorbide dinitrate, isosorbide mononitrate, and nitroglycerin). Examples of calcium channel blocking agents that may be administered in combination with the Therapeutics of the invention include, but are not limited to amlodipine, bepridil, diltiazem, felodipine, flunarizine, isradipine, nicardipine, nifedipine, nimodipine, and verapamil.

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In additional embodiments, the Therapeutics of the invention are administered in combination with other therapeutic or prophylactic regimens, such as, for example, radiation therapy.

### Example 14: Method of Treating Decreased Levels of the Polypeptide

The present invention relates to a method for treating an individual in need of an increased level of a polypeptide of the invention in the body comprising administering to such an individual a composition comprising a therapeutically effective amount of polypeptides (including agonists thereto), and/or antibodies of the invention. Moreover, it will be appreciated that conditions caused by a decrease in the standard or normal expression level of a polypeptide of the present invention in an individual may be treated by administering agonists of said polypeptide. Thus, the invention also provides a method of treatment of an individual in need of an increased level of the polypeptide comprising administering to such an individual a Therapeutic comprising an amount of the agonist (including polypeptides and antibodies of the present invention) to increase the activity level of the polypeptide in such an individual.

For example, a patient with decreased levels of a polypeptide receives a daily dose 0.1-100 ug/kg of the agonist for six consecutive days. The exact details of the dosing scheme, based on administration and formulation, are provided in Example 13.

### Example 15: Method of Treating Increased Levels of the Polypeptide

The present invention also relates to a method of treating an individual in need of a decreased level of a polypeptide of the invention in the body comprising administering to such an individual a composition comprising a therapeutically effective amount of an antagonist of the invention (including polypeptides and antibodies of the invention).

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In one example, antisense technology is used to inhibit production of a polypeptide of the present invention. This technology is one example of a method of decreasing levels of a polypeptide, due to a variety of etiologies, such as cancer.

For example, a patient diagnosed with abnormally increased levels of a polypeptide is administered intravenously antisense polynucleotides at 0.5, 1.0, 1.5, 2.0 and 3.0 mg/kg day for 21 days. This treatment is repeated after a 7-day rest period if the treatment was well tolerated. The antisense polynucleotides of the present invention can be formulated using techniques and formulations described herein (e.g. see Example 13), or otherwise known in the art.

## Example 16: Method of Treatment Using Gene Therapy-Ex Vivo

One method of gene therapy transplants fibroblasts, which are capable of expressing a polypeptide, onto a patient. Generally, fibroblasts are obtained from a subject by skin biopsy. The resulting tissue is placed in tissue-culture medium and separated into small pieces. Small chunks of the tissue are placed on a wet surface of a tissue culture flask, approximately ten pieces are placed in each flask. The flask is turned upside down, closed tight and left at room temperature over night. After 24 hours at room temperature, the flask is inverted and the chunks of tissue remain fixed to the bottom of the flask and fresh media (e.g., Ham's F12 media, with 10% FBS, penicillin and streptomycin) is added. The flasks are then incubated at 37 degree C for approximately one week.

At this time, fresh media is added and subsequently changed every several days. After an additional two weeks in culture, a monolayer of fibroblasts emerge. The monolayer is trypsinized and scaled into larger flasks.

pMV-7 (Kirschmeier, P.T. et al., DNA, 7:219-25 (1988)), flanked by the long terminal repeats of the Moloney murine sarcoma virus, is digested with EcoRI and HindIII and subsequently treated with calf intestinal phosphatase. The linear vector is fractionated on agarose gel and purified, using glass beads.

The cDNA encoding a polypeptide of the present invention can be amplified using PCR primers which correspond to the 5' and 3' end sequences respectively as set forth in Example 1 using primers and having appropriate restriction sites and initiation/stop codons, if necessary. Preferably, the 5' primer contains an EcoRI site and the 3' primer includes a HindIII site. Equal quantities of the Moloney murine sarcoma virus linear backbone and the amplified EcoRI and HindIII fragment are added together, in the presence of T4 DNA ligase. The resulting mixture is

maintained under conditions appropriate for ligation of the two fragments. The ligation mixture is then used to transform bacteria HB101, which are then plated onto agar containing kanamycin for the purpose of confirming that the vector has the gene of interest properly inserted.

The amphotropic pA317 or GP+am12 packaging cells are grown in tissue culture to confluent density in Dulbecco's Modified Eagles Medium (DMEM) with 10% calf serum (CS), penicillin and streptomycin. The MSV vector containing the gene is then added to the media and the packaging cells transduced with the vector. The packaging cells now produce infectious viral particles containing the gene (the packaging cells are now referred to as producer cells).

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Fresh media is added to the transduced producer cells, and subsequently, the media is harvested from a 10 cm plate of confluent producer cells. The spent media, containing the infectious viral particles, is filtered through a millipore filter to remove detached producer cells and this media is then used to infect fibroblast cells. Media is removed from a sub-confluent plate of fibroblasts and quickly replaced with the media from the producer cells. This media is removed and replaced with fresh media. If the titer of virus is high, then virtually all fibroblasts will be infected and no selection is required. If the titer is very low, then it is necessary to use a retroviral vector that has a selectable marker, such as neo or his. Once the fibroblasts have been efficiently infected, the fibroblasts are analyzed to determine whether protein is produced.

The engineered fibroblasts are then transplanted onto the host, either alone or after having been grown to confluence on cytodex 3 microcarrier beads.

# Example 17: Gene Therapy Using Endogenous Genes Corresponding To Polynucleotides of the Invention

Another method of gene therapy according to the present invention involves operably associating the endogenous polynucleotide sequence of the invention with a promoter via homologous recombination as described, for example, in U.S. Patent NO: 5,641,670, issued June 24, 1997; International Publication NO: WO 96/29411, published September 26, 1996; International Publication NO: WO 94/12650, published August 4, 1994; Koller et al., *Proc. Natl. Acad. Sci. USA*, 86:8932-8935 (1989); and Zijlstra et al., *Nature*, 342:435-438 (1989). This method involves the activation of a gene which is present in the target cells, but which is not expressed in the cells, or is expressed at a lower level than desired.

Polynucleotide constructs are made which contain a promoter and targeting sequences, which are homologous to the 5' non-coding sequence of endogenous polynucleotide sequence, flanking the promoter. The targeting sequence will be sufficiently near the 5' end of the polynucleotide sequence so the promoter will be operably linked to the endogenous sequence upon homologous recombination. The promoter and the targeting sequences can be amplified using PCR. Preferably, the amplified promoter contains distinct restriction enzyme sites on the 5' and 3' ends. Preferably, the 3' end of the first targeting sequence contains the same restriction enzyme

site as the 5' end of the amplified promoter and the 5' end of the second targeting sequence contains the same restriction site as the 3' end of the amplified promoter.

The amplified promoter and the amplified targeting sequences are digested with the appropriate restriction enzymes and subsequently treated with calf intestinal phosphatase. The digested promoter and digested targeting sequences are added together in the presence of T4 DNA ligase. The resulting mixture is maintained under conditions appropriate for ligation of the two fragments. The construct is size fractionated on an agarose gel, then purified by phenol extraction and ethanol precipitation.

In this Example, the polynucleotide constructs are administered as naked polynucleotides via electroporation. However, the polynucleotide constructs may also be administered with transfection-facilitating agents, such as liposomes, viral sequences, viral particles, precipitating agents, etc. Such methods of delivery are known in the art.

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Once the cells are transfected, homologous recombination will take place which results in the promoter being operably linked to the endogenous polynucleotide sequence. This results in the expression of polynucleotide corresponding to the polynucleotide in the cell. Expression may be detected by immunological staining, or any other method known in the art.

Fibroblasts are obtained from a subject by skin biopsy. The resulting tissue is placed in DMEM + 10% fetal calf serum. Exponentially growing or early stationary phase fibroblasts are trypsinized and rinsed from the plastic surface with nutrient medium. An aliquot of the cell suspension is removed for counting, and the remaining cells are subjected to centrifugation. The supernatant is aspirated and the pellet is resuspended in 5 ml of electroporation buffer (20 mM HEPES pH 7.3, 137 mM NaCl, 5 mM KCl, 0.7 mM Na₂ HPO₄, 6 mM dextrose). The cells are recentrifuged, the supernatant aspirated, and the cells resuspended in electroporation buffer containing 1 mg/ml acetylated bovine serum albumin. The final cell suspension contains approximately 3X10⁶ cells/ml. Electroporation should be performed immediately following resuspension.

Plasmid DNA is prepared according to standard techniques. For example, to construct a plasmid for targeting to the locus corresponding to the polynucleotide of the invention, plasmid pUC18 (MBI Fermentas, Amherst, NY) is digested with HindIII. The CMV promoter is amplified by PCR with an XbaI site on the 5' end and a BamHI site on the 3' end. Two non-coding sequences are amplified via PCR: one non-coding sequence (fragment 1) is amplified with a HindIII site at the 5' end and an Xba site at the 3'end; the other non-coding sequence (fragment 2) is amplified with a BamHI site at the 5'end and a HindIII site at the 3'end. The CMV promoter and the fragments (1 and 2) are digested with the appropriate enzymes (CMV promoter - XbaI and BamHI; fragment 1 - XbaI; fragment 2 - BamHI) and ligated together. The resulting ligation product is digested with HindIII, and ligated with the HindIII-digested pUC18 plasmid.

Plasmid DNA is added to a sterile cuvette with a 0.4 cm electrode gap (Bio-Rad). The final DNA concentration is generally at least  $120 \mu g/ml$ . 0.5 ml of the cell suspension (containing approximately  $1.5.X10^6$  cells) is then added to the cuvette, and the cell suspension and DNA solutions are gently mixed. Electroporation is performed with a Gene-Pulser apparatus (Bio-Rad). Capacitance and voltage are set at 960  $\mu$ F and 250-300 V, respectively. As voltage increases, cell survival decreases, but the percentage of surviving cells that stably incorporate the introduced DNA into their genome increases dramatically. Given these parameters, a pulse time of approximately 14-20 mSec should be observed.

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Electroporated cells are maintained at room temperature for approximately 5 min, and the contents of the cuvette are then gently removed with a sterile transfer pipette. The cells are added directly to 10 ml of prewarmed nutrient media (DMEM with 15% calf serum) in a 10 cm dish and incubated at 37 degree C. The following day, the media is aspirated and replaced with 10 ml of fresh media and incubated for a further 16-24 hours.

The engineered fibroblasts are then injected into the host, either alone or after having been grown to confluence on cytodex 3 microcarrier beads. The fibroblasts now produce the protein product. The fibroblasts can then be introduced into a patient as described above.

#### Example 18: Method of Treatment Using Gene Therapy - In Vivo

Another aspect of the present invention is using *in vivo* gene therapy methods to prevent, treat, and/or ameliorate hematopoietic and hematologic diseases and disorders. The gene therapy method relates to the introduction of naked nucleic acid (DNA, RNA, and antisense DNA or RNA) sequences into an animal to increase or decrease the expression of the polypeptide. The polynucleotide of the present invention may be operatively linked to (i.e., associated with) a promoter or any other genetic elements necessary for the expression of the polypeptide by the target tissue. Such gene therapy and delivery techniques and methods are known in the art, see, for example, WO90/11092, WO98/11779; U.S. Patent NO. 5693622, 5705151, 5580859; Tabata et al., Cardiovasc. Res. 35(3):470-479 (1997); Chao et al., Pharmacol. Res. 35(6):517-522 (1997); Wolff, Neuromuscul. Disord. 7(5):314-318 (1997); Schwartz et al., Gene Ther. 3(5):405-411 (1996); Tsurumi et al., Circulation 94(12):3281-3290 (1996) (incorporated herein by reference).

The polynucleotide constructs may be delivered by any method that delivers injectable materials to the cells of an animal, such as, injection into the interstitial space of tissues (heart, muscle, skin, lung, liver, intestine and the like). The polynucleotide constructs can be delivered in a pharmaceutically acceptable liquid or aqueous carrier.

The term "naked" polynucleotide, DNA or RNA, refers to sequences that are free from any delivery vehicle that acts to assist, promote, or facilitate entry into the cell, including viral sequences, viral particles, liposome formulations, lipofectin or precipitating agents and the like. However, the polynucleotides of the present invention may also be delivered in liposome

formulations (such as those taught in Felgner P.L. et al. (1995) Ann. NY Acad. Sci. 772:126-139 and Abdallah B. et al. (1995) Biol. Cell 85(1):1-7) which can be prepared by methods well known to those skilled in the art.

The polynucleotide vector constructs used in the gene therapy method are preferably constructs that will not integrate into the host genome nor will they contain sequences that allow for replication. Any strong promoter known to those skilled in the art can be used for driving the expression of DNA. Unlike other gene therapy techniques, one major advantage of introducing naked nucleic acid sequences into target cells is the transitory nature of the polynucleotide synthesis in the cells. Studies have shown that non-replicating DNA sequences can be introduced into cells to provide production of the desired polypeptide for periods of up to six months.

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The polynucleotide construct can be delivered to the interstitial space of tissues within an animal, including muscle, skin, brain, lung, liver, spleen, bone marrow, thymus, heart, lymph, blood, bone, cartilage, pancreas, kidney, gall bladder, stomach, intestine, testis, ovary, uterus, rectum, nervous system, eye, gland, and connective tissue. Interstitial space of the tissues comprises the intercellular fluid, mucopolysaccharide matrix among the reticular fibers of organ tissues, elastic fibers in the walls of vessels or chambers, collagen fibers of fibrous tissues, or that same matrix within connective tissue ensheathing muscle cells or in the lacunae of bone. It is similarly the space occupied by the plasma of the circulation and the lymph fluid of the lymphatic channels. Delivery to the interstitial space of muscle tissue is preferred for the reasons discussed below. They may be conveniently delivered by injection into the tissues comprising these cells. They are preferably delivered to and expressed in persistent, non-dividing cells which are differentiated, although delivery and expression may be achieved in non-differentiated or less completely differentiated cells, such as, for example, stem cells of blood or skin fibroblasts. *In vivo* muscle cells are particularly competent in their ability to take up and express polynucleotides.

For the naked polynucleotide injection, an effective dosage amount of DNA or RNA will be in the range of from about 0.05 g/kg body weight to about 50 mg/kg body weight. Preferably the dosage will be from about 0.005 mg/kg to about 20 mg/kg and more preferably from about 0.05 mg/kg to about 5 mg/kg. Of course, as the artisan of ordinary skill will appreciate, this dosage will vary according to the tissue site of injection. The appropriate and effective dosage of nucleic acid sequence can readily be determined by those of ordinary skill in the art and may depend on the condition being treated and the route of administration. The preferred route of administration is by the parenteral route of injection into the interstitial space of tissues. However, other parenteral routes may also be used, such as, inhalation of an aerosol formulation particularly for delivery to lungs or bronchial tissues, throat or mucous membranes of the nose. In addition, naked polynucleotide constructs can be delivered to arteries during angioplasty by the catheter used in the procedure.

The dose response effects of injected polynucleotide in muscle in vivo is determined as

follows. Suitable template DNA for production of mRNA coding for polypeptide of the present invention is prepared in accordance with a standard recombinant DNA methodology. The template DNA, which may be either circular or linear, is either used as naked DNA or complexed with liposomes. The quadriceps muscles of mice are then injected with various amounts of the template DNA.

Five to six week old female and male Balb/C mice are anesthetized by intraperitoneal injection with 0.3 ml of 2.5% Avertin. A 1.5 cm incision is made on the anterior thigh, and the quadriceps muscle is directly visualized. The template DNA is injected in 0.1 ml of carrier in a 1 cc syringe through a 27 gauge needle over one minute, approximately 0.5 cm from the distal insertion site of the muscle into the knee and about 0.2 cm deep. A suture is placed over the injection site for future localization, and the skin is closed with stainless steel clips.

After an appropriate incubation time (e.g., 7 days) muscle extracts are prepared by excising the entire quadriceps. Every fifth 15 um cross-section of the individual quadriceps muscles is histochemically stained for protein expression. A time course for protein expression may be done in a similar fashion except that quadriceps from different mice are harvested at different times. Persistence of DNA in muscle following injection may be determined by Southern blot analysis after preparing total cellular DNA and HIRT supernatants from injected and control mice. The results of the above experimentation in mice can be used to extrapolate proper dosages and other treatment parameters in humans and other animals using naked DNA.

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#### Example 19: Transgenic Animals

The polypeptides of the invention can also be expressed in transgenic animals. Animals of any species, including, but not limited to, mice, rats, rabbits, hamsters, guinea pigs, pigs, micropigs, goats, sheep, cows and non-human primates, e.g., baboons, monkeys, and chimpanzees may be used to generate transgenic animals. In a specific embodiment, techniques described herein or otherwise known in the art, are used to express polypeptides of the invention in humans, as part of a gene therapy protocol.

Any technique known in the art may be used to introduce the transgene (i.e., polynucleotides of the invention) into animals to produce the founder lines of transgenic animals. Such techniques include, but are not limited to, pronuclear microinjection (Paterson et al., Appl. Microbiol. Biotechnol. 40:691-698 (1994); Carver et al., Biotechnology (NY) 11:1263-1270 (1993); Wright et al., Biotechnology (NY) 9:830-834 (1991); and Hoppe et al., U.S. Pat. No. 4,873,191 (1989)); retrovirus mediated gene transfer into germ lines (Van der Putten et al., Proc. Natl. Acad. Sci., USA 82:6148-6152 (1985)), blastocysts or embryos; gene targeting in embryonic stem cells (Thompson et al., Cell 56:313-321 (1989)); electroporation of cells or embryos (Lo, 1983, Mol Cell. Biol. 3:1803-1814 (1983)); introduction of the polynucleotides of the invention using a gene gun (see, e.g., Ulmer et al., Science 259:1745 (1993); introducing nucleic acid

constructs into embryonic pleuripotent stem cells and transferring the stem cells back into the blastocyst; and sperm-mediated gene transfer (Lavitrano et al., Cell 57:717-723 (1989); etc. For a review of such techniques, see Gordon, "Transgenic Animals," Intl. Rev. Cytol. 115:171-229 (1989), which is incorporated by reference herein in its entirety.

Any technique known in the art may be used to produce transgenic clones containing polynucleotides of the invention, for example, nuclear transfer into enucleated oocytes of nuclei from cultured embryonic, fetal, or adult cells induced to quiescence (Campell et al., Nature 380:64-66 (1996); Wilmut et al., Nature 385:810-813 (1997)).

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The present invention provides for transgenic animals that carry the transgene in all their cells, as well as animals which carry the transgene in some, but not all their cells, i.e., mosaic animals or chimeric. The transgene may be integrated as a single transgene or as multiple copies such as in concatamers, e.g., head-to-head tandems or head-to-tail tandems. The transgene may also be selectively introduced into and activated in a particular cell type by following, for example, the teaching of Lasko et al. (Lasko et al., Proc. Natl. Acad. Sci. USA 89:6232-6236 (1992)). The regulatory sequences required for such a cell-type specific activation will depend upon the particular cell type of interest, and will be apparent to those of skill in the art. When it is desired that the polynucleotide transgene be integrated into the chromosomal site of the endogenous gene, gene targeting is preferred. Briefly, when such a technique is to be utilized, vectors containing some nucleotide sequences homologous to the endogenous gene are designed for the purpose of integrating, via homologous recombination with chromosomal sequences, into and disrupting the function of the nucleotide sequence of the endogenous gene. The transgene may also be selectively introduced into a particular cell type, thus inactivating the endogenous gene in only that cell type, by following, for example, the teaching of Gu et al., Gu et al., Science 265:103-106 (1994)). The regulatory sequences required for such a cell-type specific inactivation will depend upon the particular cell type of interest, and will be apparent to those of skill in the art.

Once transgenic animals have been generated, the expression of the recombinant gene may be assayed utilizing standard techniques. Initial screening may be accomplished by Southern blot analysis or PCR techniques to analyze animal tissues to verify that integration of the transgene has taken place. The level of mRNA expression of the transgene in the tissues of the transgenic animals may also be assessed using techniques which include, but are not limited to, Northern blot analysis of tissue samples obtained from the animal, in situ hybridization analysis, and reverse transcriptase-PCR (rt-PCR). Samples of transgenic gene-expressing tissue may also be evaluated immunocytochemically or immunohistochemically using antibodies specific for the transgene product.

Once the founder animals are produced, they may be bred, inbred, outbred, or crossbred to produce colonies of the particular animal. Examples of such breeding strategies include, but are not limited to: outbreeding of founder animals with more than one integration site in order to

establish separate lines; inbreeding of separate lines in order to produce compound transgenics that express the transgene at higher levels because of the effects of additive expression of each transgene; crossing of heterozygous transgenic animals to produce animals homozygous for a given integration site in order to both augment expression and eliminate the need for screening of animals by DNA analysis; crossing of separate homozygous lines to produce compound heterozygous or homozygous lines; and breeding to place the transgene on a distinct background that is appropriate for an experimental model of interest.

Transgenic animals of the invention have uses which include, but are not limited to, animal model systems useful in elaborating the biological function of polypeptides of the present invention, studying conditions and/or disorders associated with aberrant expression, and in screening for compounds effective in ameliorating such conditions and/or disorders.

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### Example 20: Knock-Out Animals

Endogenous gene expression can also be reduced by inactivating or "knocking out" the gene and/or its promoter using targeted homologous recombination. (e.g., see Smithies et al., Nature 317:230-234 (1985); Thomas & Capecchi, Cell 51:503-512 (1987); Thompson et al., Cell 5:313-321 (1989); each of which is incorporated by reference herein in its entirety). For example, a mutant, non-functional polynucleotide of the invention (or a completely unrelated DNA sequence) flanked by DNA homologous to the endogenous polynucleotide sequence (either the coding regions or regulatory regions of the gene) can be used, with or without a selectable marker and/or a negative selectable marker, to transfect cells that express polypeptides of the invention in vivo. In another embodiment, techniques known in the art are used to generate knockouts in cells that contain, but do not express the gene of interest. Insertion of the DNA construct, via targeted homologous recombination, results in inactivation of the targeted gene. Such approaches are particularly suited in research and agricultural fields where modifications to embryonic stem cells can be used to generate animal offspring with an inactive targeted gene (e.g., see Thomas & Capecchi 1987 and Thompson 1989, supra). However this approach can be routinely adapted for use in humans provided the recombinant DNA constructs are directly administered or targeted to the required site in vivo using appropriate viral vectors that will be apparent to those of skill in the art.

In further embodiments of the invention, cells that are genetically engineered to express the polypeptides of the invention, or alternatively, that are genetically engineered not to express the polypeptides of the invention (e.g., knockouts) are administered to a patient in vivo. Such cells may be obtained from the patient (i.e., animal, including human) or an MHC compatible donor and can include, but are not limited to fibroblasts, bone marrow cells, blood cells (e.g., lymphocytes), adipocytes, muscle cells, endothelial cells etc. The cells are genetically engineered in vitro using recombinant DNA techniques to introduce the coding sequence of polypeptides of the invention

into the cells, or alternatively, to disrupt the coding sequence and/or endogenous regulatory sequence associated with the polypeptides of the invention, e.g., by transduction (using viral vectors, and preferably vectors that integrate the transgene into the cell genome) or transfection procedures, including, but not limited to, the use of plasmids, cosmids, YACs, naked DNA, electroporation, liposomes, etc. The coding sequence of the polypeptides of the invention can be placed under the control of a strong constitutive or inducible promoter or promoter/enhancer to achieve expression, and preferably secretion, of the polypeptides of the invention. The engineered cells which express and preferably secrete the polypeptides of the invention can be introduced into the patient systemically, e.g., in the circulation, or intraperitoneally.

Alternatively, the cells can be incorporated into a matrix and implanted in the body, e.g., genetically engineered fibroblasts can be implanted as part of a skin graft; genetically engineered endothelial cells can be implanted as part of a lymphatic or vascular graft. (See, for example, Anderson et al. U.S. Patent No. 5,399,349; and Mulligan & Wilson, U.S. Patent No. 5,460,959 each of which is incorporated by reference herein in its entirety).

When the cells to be administered are non-autologous or non-MHC compatible cells, they can be administered using well known techniques which prevent the development of a host immune response against the introduced cells. For example, the cells may be introduced in an encapsulated form which, while allowing for an exchange of components with the immediate extracellular environment, does not allow the introduced cells to be recognized by the host immune system.

Transgenic and "knock-out" animals of the invention have uses which include, but are not limited to, animal model systems useful in elaborating the biological function of polypeptides of the present invention, studying conditions and/or disorders associated with aberrant expression, and in screening for compounds effective in ameliorating such conditions and/or disorders.

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# Example 21: Assays Detecting Stimulation or Inhibition of B cell Proliferation and Differentiation

Generation of functional humoral immune responses requires both soluble and cognate signaling between B-lineage cells and their microenvironment. Signals may impart a positive stimulus that allows a B-lineage cell to continue its programmed development, or a negative stimulus that instructs the cell to arrest its current developmental pathway. To date, numerous stimulatory and inhibitory signals have been found to influence B cell responsiveness including IL-2, IL-4, IL-5, IL-6, IL-7, IL10, IL-13, IL-14 and IL-15. Interestingly, these signals are by themselves weak effectors but can, in combination with various co-stimulatory proteins, induce activation, proliferation, differentiation, homing, tolerance and death among B cell populations.

One of the best studied classes of B-cell co-stimulatory proteins is the TNF-superfamily. Within this family CD40, CD27, and CD30 along with their respective ligands CD154, CD70, and

CD153 have been found to regulate a variety of immune responses. Assays which allow for the detection and/or observation of the proliferation and differentiation of these B-cell populations and their precursors are valuable tools in determining the effects various proteins may have on these B-cell populations in terms of proliferation and differentiation. Listed below are two assays designed to allow for the detection of the differentiation, proliferation, or inhibition of B-cell populations and their precursors.

In Vitro Assay- Agonists or antagonists of the invention can be assessed for its ability to induce activation, proliferation, differentiation or inhibition and/or death in B-cell populations and their precursors. The activity of the agonists or antagonists of the invention on purified human tonsillar B cells, measured qualitatively over the dose range from 0.1 to 10,000 ng/mL, is assessed in a standard B-lymphocyte co-stimulation assay in which purified tonsillar B cells are cultured in the presence of either formalin-fixed *Staphylococcus aureus* Cowan I (SAC) or immobilized anti-human IgM antibody as the priming agent. Second signals such as IL-2 and IL-15 synergize with SAC and IgM crosslinking to elicit B cell proliferation as measured by tritiated-thymidine incorporation. Novel synergizing agents can be readily identified using this assay. The assay involves isolating human tonsillar B cells by magnetic bead (MACS) depletion of CD3-positive cells. The resulting cell population is greater than 95% B cells as assessed by expression of CD45R(B220).

Various dilutions of each sample are placed into individual wells of a 96-well plate to which are added 10⁵ B-cells suspended in culture medium (RPMI 1640 containing 10% FBS, 5 X 10⁻⁵M 2ME, 100U/ml penicillin, 10ug/ml streptomycin, and 10⁻⁵ dilution of SAC) in a total volume of 150ul. Proliferation or inhibition is quantitated by a 20h pulse (1uCi/well) with 3H-thymidine (6.7 Ci/mM) beginning 72h post factor addition. The positive and negative controls are IL2 and medium respectively.

In vivo Assay- BALB/c mice are injected (i.p.) twice per day with buffer only, or 2 mg/Kg of agonists or antagonists of the invention, or truncated forms thereof. Mice receive this treatment for 4 consecutive days, at which time they are sacrificed and various tissues and serum collected for analyses. Comparison of H&E sections from normal spleens and spleens treated with agonists or antagonists of the invention identify the results of the activity of the agonists or antagonists on spleen cells, such as the diffusion of peri-arterial lymphatic sheaths, and/or significant increases in the nucleated cellularity of the red pulp regions, which may indicate the activation of the differentiation and proliferation of B-cell populations. Immunohistochemical studies using a B cell marker, anti-CD45R(B220), are used to determine whether any physiological changes to splenic cells, such as splenic disorganization, are due to increased B-cell representation within loosely defined B-cell zones that infiltrate established T-cell regions.

Flow cytometric analyses of the spleens from mice treated with agonist or antagonist is used to indicate whether the agonists or antagonists specifically increases the proportion of ThB+, CD45R(B220)dull B cells over that which is observed in control mice.

Likewise, a predicted consequence of increased mature B-cell representation *in vivo* is a relative increase in serum Ig titers. Accordingly, serum IgM and IgA levels are compared between buffer and agonists or antagonists-treated mice.

The studies described in this example tested activity of agonists or antagonists of the invention. However, one skilled in the art could easily modify the exemplified studies to test the activity of polynucleotides or polypeptides of the invention (e.g., gene therapy).

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### Example 22: T Cell Proliferation Assay

A CD3-induced proliferation assay is performed on PBMCs and is measured by the uptake of ³H-thymidine. The assay is performed as follows. Ninety-six well plates are coated with 100 ul/well of mAb to CD3 (HIT3a, Pharmingen) or isotype-matched control mAb (B33.1) overnight at 4 degrees C (1 µg/ml in .05M bicarbonate buffer, pH 9.5), then washed three times with PBS. PBMC are isolated by F/H gradient centrifugation from human peripheral blood and added to quadruplicate wells (5 x 10⁴/well) of mAb coated plates in RPMI containing 10% FCS and P/S in the presence of varying concentrations of agonists or antagonists of the invention (total volume 200 ul). Relevant protein buffer and medium alone are controls. After 48 hr. culture at 37 degrees C, plates are spun for 2 min. at 1000 rpm and 100 µl of supernatant is removed and stored -20 degrees C for measurement of IL-2 (or other cytokines) if effect on proliferation is observed. Wells are supplemented with 100 ul of medium containing 0.5 uCi of ³H-thymidine and cultured at 37 degrees C for 18-24 hr. Wells are harvested and incorporation of ³H-thymidine used as a measure of proliferation. Anti-CD3 alone is the positive control for proliferation. IL-2 (100 U/ml) is also used as a control which enhances proliferation. Control antibody which does not induce proliferation of T cells is used as the negative control for the effects of agonists or antagonists of the invention.

The studies described in this example tested activity of agonists or antagonists of the invention. However, one skilled in the art could easily modify the exemplified studies to test the activity of polynucleotides or polypeptides of the invention (e.g., gene therapy).

# Example 23: Effect of Agonists or Antagonists of the Invention on the Expression of MHC Class II, Costimulatory and Adhesion Molecules and Cell Differentiation of Monocytes and Monocyte-Derived Human Dendritic Cells

Dendritic cells are generated by the expansion of proliferating precursors found in the peripheral blood: adherent PBMC or elutriated monocytic fractions are cultured for 7-10 days with

GM-CSF (50 ng/ml) and IL-4 (20 ng/ml). These dendritic cells have the characteristic phenotype of immature cells (expression of CD1, CD80, CD86, CD40 and MHC class II antigens). Treatment with activating factors, such as TNF- $\alpha$ , causes a rapid change in surface phenotype (increased expression of MHC class I and II, costimulatory and adhesion molecules, downregulation of FCYRII, upregulation of CD83). These changes correlate with increased antigen-presenting capacity and with functional maturation of the dendritic cells.

FACS analysis of surface antigens is performed as follows. Cells are treated 1-3 days with increasing concentrations of agonist or antagonist of the invention or LPS (positive control), washed with PBS containing 1% BSA and 0.02 mM sodium azide, and then incubated with 1:20 dilution of appropriate FITC- or PE-labeled monoclonal antibodies for 30 minutes at 4 degrees C. After an additional wash, the labeled cells are analyzed by flow cytometry on a FACScan (Becton Dickinson).

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Effect on the production of cytokines. Cytokines generated by dendritic cells, in particular IL-12, are important in the initiation of T-cell dependent immune responses. IL-12 strongly influences the development of Thl helper T-cell immune response, and induces cytotoxic T and NK cell function. An ELISA is used to measure the IL-12 release as follows. Dendritic cells (10⁶/ml) are treated with increasing concentrations of agonists or antagonists of the invention for 24 hours. LPS (100 ng/ml) is added to the cell culture as positive control. Supernatants from the cell cultures are then collected and analyzed for IL-12 content using commercial ELISA kit (e.g., R & D Systems (Minneapolis, MN)). The standard protocols provided with the kits are used.

Effect on the expression of MHC Class II, costimulatory and adhesion molecules. Three major families of cell surface antigens can be identified on monocytes: adhesion molecules, molecules involved in antigen presentation, and Fc receptor. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result in changes in the antigen presenting capacity of monocytes and ability to induce T cell activation. Increased expression of Fc receptors may correlate with improved monocyte cytotoxic activity, cytokine release and phagocytosis.

FACS analysis is used to examine the surface antigens as follows. Monocytes are treated 1-5 days with increasing concentrations of agonists or antagonists of the invention or LPS (positive control), washed with PBS containing 1% BSA and 0.02 mM sodium azide, and then incubated with 1:20 dilution of appropriate FITC- or PE-labeled monoclonal antibodies for 30 minutes at 4 degrees C. After an additional wash, the labeled cells are analyzed by flow cytometry on a FACScan (Becton Dickinson).

Monocyte activation and/or increased survival. Assays for molecules that activate (or alternatively, inactivate) monocytes and/or increase monocyte survival (or alternatively, decrease monocyte survival) are known in the art and may routinely be applied to determine whether a

molecule of the invention functions as an inhibitor or activator of monocytes. Agonists or antagonists of the invention can be screened using the three assays described below. For each of these assays, Peripheral blood mononuclear cells (PBMC) are purified from single donor leukopacks (American Red Cross, Baltimore, MD) by centrifugation through a Histopaque gradient (Sigma). Monocytes are isolated from PBMC by counterflow centrifugal elutriation.

Monocyte Survival Assay. Human peripheral blood monocytes progressively lose viability when cultured in absence of serum or other stimuli. Their death results from internally regulated processes (apoptosis). Addition to the culture of activating factors, such as TNF-alpha dramatically improves cell survival and prevents DNA fragmentation. Propidium iodide (PI) staining is used to measure apoptosis as follows. Monocytes are cultured for 48 hours in polypropylene tubes in serum-free medium (positive control), in the presence of 100 ng/ml TNF-alpha (negative control), and in the presence of varying concentrations of the compound to be tested. Cells are suspended at a concentration of 2 x 10⁶/ml in PBS containing PI at a final concentration of 5 μg/ml, and then incubated at room temperature for 5 minutes before FACScan analysis. PI uptake has been demonstrated to correlate with DNA fragmentation in this experimental paradigm.

Effect on cytokine release. An important function of monocytes/macrophages is their regulatory activity on other cellular populations of the immune system through the release of cytokines after stimulation. An ELISA to measure cytokine release is performed as follows. Human monocytes are incubated at a density of  $5 \times 10^5$  cells/ml with increasing concentrations of agonists or antagonists of the invention and under the same conditions, but in the absence of agonists or antagonists. For IL-12 production, the cells are primed overnight with IFN (100 U/ml) in the presence of agonist or antagonist of the invention. LPS (10 ng/ml) is then added. Conditioned media are collected after 24h and kept frozen until use. Measurement of TNF-alpha, IL-10, MCP-1 and IL-8 is then performed using a commercially available ELISA kit (e.g., R & D Systems (Minneapolis, MN)) and applying the standard protocols provided with the kit.

Oxidative burst. Purified monocytes are plated in 96-w plate at  $2-1\times10^5$  cell/well. Increasing concentrations of agonists or antagonists of the invention are added to the wells in a total volume of 0.2 ml culture medium (RPMI 1640 + 10% FCS, glutamine and antibiotics). After 3 days incubation, the plates are centrifuged and the medium is removed from the wells. To the macrophage monolayers, 0.2 ml per well of phenol red solution (140 mM NaCl, 10 mM potassium phosphate buffer pH 7.0, 5.5 mM dextrose, 0.56 mM phenol red and 19 U/ml of HRPO) is added, together with the stimulant (200 nM PMA). The plates are incubated at 37°C for 2 hours and the reaction is stopped by adding 20  $\mu$ l 1N NaOH per well. The absorbance is read at 610 nm. To calculate the amount of  $H_2O_2$  produced by the macrophages, a standard curve of a  $H_2O_2$  solution of known molarity is performed for each experiment.

The studies described in this example tested activity of agonists or antagonists of the invention. However, one skilled in the art could easily modify the exemplified studies to test the activity of polynucleotides or polypeptides of the invention (e.g., gene therapy).

# Example 24: Diabetic Mouse and Glucocorticoid-Impaired Wound Healing Models

Diabetic db+/db+ Mouse Model.

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To demonstrate that an agonist or antagonist of the invention accelerates the healing process, the genetically diabetic mouse model of wound healing is used. The full thickness wound healing model in the db+/db+ mouse is a well characterized, clinically relevant and reproducible model of impaired wound healing. Healing of the diabetic wound is dependent on formation of granulation tissue and re-epithelialization rather than contraction (Gartner, M.H. et al., J. Surg. Res. 52:389 (1992); Greenhalgh, D.G. et al., Am. J. Pathol. 136:1235 (1990)).

The diabetic animals have many of the characteristic features observed in Type II diabetes mellitus. Homozygous (db+/db+) mice are obese in comparison to their normal heterozygous (db+/+m) littermates. Mutant diabetic (db+/db+) mice have a single autosomal recessive mutation on chromosome 4 (db+) (Coleman et al. Proc. Natl. Acad. Sci. USA 77:283-293 (1982)). Animals show polyphagia, polydipsia and polyuria. Mutant diabetic mice (db+/db+) have elevated blood glucose, increased or normal insulin levels, and suppressed cell-mediated immunity (Mandel et al., J. Immunol. 120:1375 (1978); Debray-Sachs, M. et al., Clin. Exp. Immunol. 51(1):1-7 (1983); Leiter et al., Am. J. of Pathol. 114:46-55 (1985)). Peripheral neuropathy, myocardial complications, and microvascular lesions, basement membrane thickening and glomerular filtration abnormalities have been described in these animals (Norido, F. et al., Exp. Neurol. 83(2):221-232 (1984); Robertson et al., Diabetes 29(1):60-67 (1980); Giacomelli et al., Lab Invest. 40(4):460-473 (1979); Coleman, D.L., Diabetes 31 (Suppl):1-6 (1982)). These homozygous diabetic mice develop hyperglycemia that is resistant to insulin analogous to human type II diabetes (Mandel et al., J. Immunol. 120:1375-1377 (1978)).

The characteristics observed in these animals suggests that healing in this model may be similar to the healing observed in human diabetes (Greenhalgh, et al., Am. J. of Pathol. 136:1235-1246 (1990)).

Genetically diabetic female C57BL/KsJ (db+/db+) mice and their non-diabetic (db+/+m) heterozygous littermates are used in this study (Jackson Laboratories). The animals are purchased at 6 weeks of age and are 8 weeks old at the beginning of the study. Animals are individually housed and received food and water ad libitum. All manipulations are performed using aseptic techniques. The experiments are conducted according to the rules and guidelines of Human Genome Sciences, Inc. Institutional Animal Care and Use Committee and the Guidelines for the Care and Use of Laboratory Animals.

Wounding protocol is performed according to previously reported methods (Tsuboi, R. and Rifkin, D.B., J. Exp. Med. 172:245-251 (1990)). Briefly, on the day of wounding, animals are anesthetized with an intraperitoneal injection of Avertin (0.01 mg/mL), 2,2,2-tribromoethanol and 2-methyl-2-butanol dissolved in deionized water. The dorsal region of the animal is shaved and the skin washed with 70% ethanol solution and iodine. The surgical area is dried with sterile gauze prior to wounding. An 8 mm full-thickness wound is then created using a Keyes tissue punch. Immediately following wounding, the surrounding skin is gently stretched to eliminate wound expansion. The wounds are left open for the duration of the experiment. Application of the treatment is given topically for 5 consecutive days commencing on the day of wounding. Prior to treatment, wounds are gently cleansed with sterile saline and gauze sponges.

Wounds are visually examined and photographed at a fixed distance at the day of surgery and at two day intervals thereafter. Wound closure is determined by daily measurement on days 1-5 and on day 8. Wounds are measured horizontally and vertically using a calibrated Jameson caliper. Wounds are considered healed if granulation tissue is no longer visible and the wound is covered by a continuous epithelium.

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An agonist or antagonist of the invention is administered using at a range different doses, from 4mg to 500mg per wound per day for 8 days in vehicle. Vehicle control groups received 50mL of vehicle solution.

Animals are euthanized on day 8 with an intraperitoneal injection of sodium pentobarbital (300mg/kg). The wounds and surrounding skin are then harvested for histology and immunohistochemistry. Tissue specimens are placed in 10% neutral buffered formalin in tissue cassettes between biopsy sponges for further processing.

Three groups of 10 animals each (5 diabetic and 5 non-diabetic controls) are evaluated: 1) Vehicle placebo control, 2) untreated group, and 3) treated group.

Wound closure is analyzed by measuring the area in the vertical and horizontal axis and obtaining the total square area of the wound. Contraction is then estimated by establishing the differences between the initial wound area (day 0) and that of post treatment (day 8). The wound area on day 1 is 64mm², the corresponding size of the dermal punch. Calculations are made using the following formula:

[Open area on day 8] - [Open area on day 1] / [Open area on day 1]

Specimens are fixed in 10% buffered formalin and paraffin embedded blocks are sectioned perpendicular to the wound surface (5mm) and cut using a Reichert-Jung microtome. Routine hematoxylin-eosin (H&E) staining is performed on cross-sections of bisected wounds. Histologic examination of the wounds are used to assess whether the healing process and the morphologic appearance of the repaired skin is altered by treatment with an agonist or antagonist of the invention.

This assessment included verification of the presence of cell accumulation, inflammatory cells, capillaries, fibroblasts, re-epithelialization and epidermal maturity (Greenhalgh, D.G. et al., Am. J. Pathol. 136:1235 (1990)). A calibrated lens micrometer is used by a blinded observer.

Tissue sections are also stained immunohistochemically with a polyclonal rabbit anti-human keratin antibody using ABC Elite detection system. Human skin is used as a positive tissue control while non-immune IgG is used as a negative control. Keratinocyte growth is determined by evaluating the extent of reepithelialization of the wound using a calibrated lens micrometer.

Proliferating cell nuclear antigen/cyclin (PCNA) in skin specimens is demonstrated by using anti-PCNA antibody (1:50) with an ABC Elite detection system. Human colon cancer served as a positive tissue control and human brain tissue is used as a negative tissue control. Each specimen included a section with omission of the primary antibody and substitution with non-immune mouse IgG. Ranking of these sections is based on the extent of proliferation on a scale of 0-8, the lower side of the scale reflecting slight proliferation to the higher side reflecting intense proliferation.

Experimental data are analyzed using an unpaired t test. A p value of < 0.05 is considered significant.

#### Steroid Impaired Rat Model

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The inhibition of wound healing by steroids has been well documented in various in vitro and in vivo systems (Wahl, Glucocorticoids and Wound healing. In: Anti-Inflammatory Steroid Action: Basic and Clinical Aspects. 280-302 (1989); Wahlet al., J. Immunol. 115: 476-481 (1975); Werb et al., J. Exp. Med. 147:1684-1694 (1978)). Glucocorticoids retard wound healing by inhibiting angiogenesis, decreasing vascular permeability (Ebert et al., An. Intern. Med. 37:701-705 (1952)), fibroblast proliferation, and collagen synthesis (Beck et al., Growth Factors. 5: 295-304 (1991); Haynes et al., J. Clin. Invest. 61: 703-797 (1978)) and producing a transient reduction of circulating monocytes (Haynes et al., J. Clin. Invest. 61: 703-797 (1978); Wahl, "Glucocorticoids and wound healing", In: Antiinflammatory Steroid Action: Basic and Clinical Aspects, Academic Press, New York, pp. 280-302 (1989)). The systemic administration of steroids to impaired wound healing is a well establish phenomenon in rats (Beck et al., Growth Factors. 5: 295-304 (1991); Haynes et al., J. Clin. Invest. 61: 703-797 (1978); Wahl, "Glucocorticoids and wound healing", In: Antiinflammatory Steroid Action: Basic and Clinical Aspects, Academic Press, New York, pp. 280-302 (1989); Pierce et al., Proc. Natl. Acad. Sci. USA 86: 2229-2233 (1989)).

To demonstrate that an agonist or antagonist of the invention can accelerate the healing process, the effects of multiple topical applications of the agonist or antagonist on full thickness excisional skin wounds in rats in which healing has been impaired by the systemic administration of methylprednisolone is assessed.

Young adult male Sprague Dawley rats weighing 250-300 g (Charles River Laboratories) are used in this example. The animals are purchased at 8 weeks of age and are 9 weeks old at the

beginning of the study. The healing response of rats is impaired by the systemic administration of methylprednisolone (17mg/kg/rat intramuscularly) at the time of wounding. Animals are individually housed and received food and water ad libitum. All manipulations are performed using aseptic techniques. This study is conducted according to the rules and guidelines of Human Genome Sciences, Inc. Institutional Animal Care and Use Committee and the Guidelines for the Care and Use of Laboratory Animals.

The wounding protocol is followed according to section A, above. On the day of wounding, animals are anesthetized with an intramuscular injection of ketamine (50 mg/kg) and xylazine (5 mg/kg). The dorsal region of the animal is shaved and the skin washed with 70% ethanol and iodine solutions. The surgical area is dried with sterile gauze prior to wounding. An 8 mm full-thickness wound is created using a Keyes tissue punch. The wounds are left open for the duration of the experiment. Applications of the testing materials are given topically once a day for 7 consecutive days commencing on the day of wounding and subsequent to methylprednisolone administration. Prior to treatment, wounds are gently cleansed with sterile saline and gauze sponges.

Wounds are visually examined and photographed at a fixed distance at the day of wounding and at the end of treatment. Wound closure is determined by daily measurement on days 1-5 and on day 8. Wounds are measured horizontally and vertically using a calibrated Jameson caliper. Wounds are considered healed if granulation tissue is no longer visible and the wound is covered by a continuous epithelium.

The agonist or antagonist of the invention is administered using at a range different doses, from 4mg to 500mg per wound per day for 8 days in vehicle. Vehicle control groups received 50mL of vehicle solution.

Animals are euthanized on day 8 with an intraperitoneal injection of sodium pentobarbital (300mg/kg). The wounds and surrounding skin are then harvested for histology. Tissue specimens are placed in 10% neutral buffered formalin in tissue cassettes between biopsy sponges for further processing.

Three groups of 10 animals each (5 with methylprednisolone and 5 without glucocorticoid) are evaluated: 1) Untreated group 2) Vehicle placebo control 3) treated groups.

Wound closure is analyzed by measuring the area in the vertical and horizontal axis and obtaining the total area of the wound. Closure is then estimated by establishing the differences between the initial wound area (day 0) and that of post treatment (day 8). The wound area on day 1 is 64mm², the corresponding size of the dermal punch. Calculations are made using the following formula:

[Open area on day 8] - [Open area on day 1] / [Open area on day 1]

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Specimens are fixed in 10% buffered formalin and paraffin embedded blocks are sectioned perpendicular to the wound surface (5mm) and cut using an Olympus microtome. Routine hematoxylin-

eosin (H&E) staining is performed on cross-sections of bisected wounds. Histologic examination of the wounds allows assessment of whether the healing process and the morphologic appearance of the repaired skin is improved by treatment with an agonist or antagonist of the invention. A calibrated lens micrometer is used by a blinded observer to determine the distance of the wound gap.

Experimental data are analyzed using an unpaired t test. A p value of < 0.05 is considered significant.

The studies described in this example tested activity of agonists or antagonists of the invention. However, one skilled in the art could easily modify the exemplified studies to test the activity of polynucleotides or polypeptides of the invention (e.g., gene therapy).

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### Example 25: Lymphadema Animal Model

The purpose of this experimental approach is to create an appropriate and consistent lymphedema model for testing the therapeutic effects of an agonist or antagonist of the invention in lymphangiogenesis and re-establishment of the lymphatic circulatory system in the rat hind limb. Effectiveness is measured by swelling volume of the affected limb, quantification of the amount of lymphatic vasculature, total blood plasma protein, and histopathology. Acute lymphedema is observed for 7-10 days. Perhaps more importantly, the chronic progress of the edema is followed for up to 3-4 weeks.

Prior to beginning surgery, blood sample is drawn for protein concentration analysis. Male rats weighing approximately ~350g are dosed with Pentobarbital. Subsequently, the right legs are shaved from knee to hip. The shaved area is swabbed with gauze soaked in 70% EtOH. Blood is drawn for serum total protein testing. Circumference and volumetric measurements are made prior to injecting dye into paws after marking 2 measurement levels (0.5 cm above heel, at mid-pt of dorsal paw). The intradermal dorsum of both right and left paws are injected with 0.05 ml of 1% Evan's Blue. Circumference and volumetric measurements are then made following injection of dye into paws.

Using the knee joint as a landmark, a mid-leg inguinal incision is made circumferentially allowing the femoral vessels to be located. Forceps and hemostats are used to dissect and separate the skin flaps. After locating the femoral vessels, the lymphatic vessel that runs along side and underneath the vessel(s) is located. The main lymphatic vessels in this area are then electrically coagulated or suture ligated.

Using a microscope, muscles in back of the leg (near the semitendinosis and adductors) are bluntly dissected. The popliteal lymph node is then located. The 2 proximal and 2 distal lymphatic vessels and distal blood supply of the popliteal node are then ligated by suturing. The popliteal lymph node, and any accompanying adipose tissue, is then removed by cutting connective tissues.

Care is taken to control any mild bleeding resulting from this procedure. After lymphatics are occluded, the skin flaps are sealed by using liquid skin (Vetbond) (AJ Buck). The separated skin edges

are sealed to the underlying muscle tissue while leaving a gap of ~0.5 cm around the leg. Skin also may be anchored by suturing to underlying muscle when necessary.

To avoid infection, animals are housed individually with mesh (no bedding). Recovering animals are checked daily through the optimal edematous peak, which typically occurred by day 5-7. The plateau edematous peak are then observed. To evaluate the intensity of the lymphedema, the circumference and volumes of 2 designated places on each paw before operation and daily for 7 days are measured. The effect of plasma proteins on lymphedema is determined and whether protein analysis is a useful testing perimeter is also investigated. The weights of both control and edematous limbs are evaluated at 2 places. Analysis is performed in a blind manner.

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Circumference Measurements: Under brief gas anesthetic to prevent limb movement, a cloth tape is used to measure limb circumference. Measurements are done at the ankle bone and dorsal paw by 2 different people and those 2 readings are averaged. Readings are taken from both control and edematous limbs.

Volumetric Measurements: On the day of surgery, animals are anesthetized with Pentobarbital and are tested prior to surgery. For daily volumetrics animals are under brief halothane anesthetic (rapid immobilization and quick recovery), and both legs are shaved and equally marked using waterproof marker on legs. Legs are first dipped in water, then dipped into instrument to each marked level then measured by Buxco edema software(Chen/Victor). Data is recorded by one person, while the other is dipping the limb to marked area.

Blood-plasma protein measurements: Blood is drawn, spun, and serum separated prior to surgery and then at conclusion for total protein and Ca2⁺ comparison.

Limb Weight Comparison: After drawing blood, the animal is prepared for tissue collection. The limbs are amputated using a quillitine, then both experimental and control legs are cut at the ligature and weighed. A second weighing is done as the tibio-cacaneal joint is disarticulated and the foot is weighed.

Histological Preparations: The transverse muscle located behind the knee (popliteal) area is dissected and arranged in a metal mold, filled with freezeGel, dipped into cold methylbutane, placed into labeled sample bags at - 80EC until sectioning. Upon sectioning, the muscle is observed under fluorescent microscopy for lymphatics..

The studies described in this example tested activity of agonists or antagonists of the invention. However, one skilled in the art could easily modify the exemplified studies to test the activity of polynucleotides or polypeptides of the invention (e.g., gene therapy).

# Example 26: Suppression of TNF alpha-induced adhesion molecule expression by an Agonist of the Invention

The recruitment of lymphocytes to areas of inflammation and angiogenesis involves specific receptor-ligand interactions between cell surface adhesion molecules (CAMs) on lymphocytes and the

vascular endothelium. The adhesion process, in both normal and pathological settings, follows a multistep cascade that involves intercellular adhesion molecule-1 (ICAM-1), vascular cell adhesion molecule-1 (VCAM-1), and endothelial leukocyte adhesion molecule-1 (E-selectin) expression on endothelial cells (EC). The expression of these molecules and others on the vascular endothelium determines the efficiency with which leukocytes may adhere to the local vasculature and extravasate into the local tissue during the development of an inflammatory response. The local concentration of cytokines and growth factor participate in the modulation of the expression of these CAMs.

Tumor necrosis factor alpha (TNF-a), a potent proinflammatory cytokine, is a stimulator of all three CAMs on endothelial cells and may be involved in a wide variety of inflammatory responses, often resulting in a pathological outcome.

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The potential of an agonist or antagonist of the invention to mediate a suppression of TNF-a induced CAM expression can be examined. A modified ELISA assay which uses ECs as a solid phase absorbent is employed to measure the amount of CAM expression on TNF-a treated ECs when costimulated with a member of the FGF family of proteins.

To perform the experiment, human umbilical vein endothelial cell (HUVEC) cultures are obtained from pooled cord harvests and maintained in growth medium (EGM-2; Clonetics, San Diego, CA) supplemented with 10% FCS and 1% penicillin/streptomycin in a 37 degree C humidified incubator containing 5% CO₂. HUVECs are seeded in 96-well plates at concentrations of 1 x 10⁴ cells/well in EGM medium at 37 degree C for 18-24 hrs or until confluent. The monolayers are subsequently washed 3 times with a serum-free solution of RPMI-1640 supplemented with 100 U/ml penicillin and 100 mg/ml streptomycin, and treated with a given cytokine and/or growth factor(s) for 24 h at 37 degree C. Following incubation, the cells are then evaluated for CAM expression.

Human Umbilical Vein Endothelial cells (HUVECs) are grown in a standard 96 well plate to confluence. Growth medium is removed from the cells and replaced with 90 ul of 199 Medium (10% FBS). Samples for testing and positive or negative controls are added to the plate in triplicate (in 10 ul volumes). Plates are incubated at 37 degree C for either 5 h (selectin and integrin expression) or 24 h (integrin expression only). Plates are aspirated to remove medium and  $100 \mu l$  of 0.1% paraformaldehyde-PBS(with Ca++ and Mg++) is added to each well. Plates are held at 4°C for 30 min.

Fixative is then removed from the wells and wells are washed 1X with PBS(+Ca,Mg)+0.5% BSA and drained. Do not allow the wells to dry. Add 10  $\mu$ l of diluted primary antibody to the test and control wells. Anti-ICAM-1-Biotin, Anti-VCAM-1-Biotin and Anti-E-selectin-Biotin are used at a concentration of 10  $\mu$ g/ml (1:10 dilution of 0.1 mg/ml stock antibody). Cells are incubated at 37°C for 30 min. in a humidified environment. Wells are washed X3 with PBS(+Ca,Mg)+0.5% BSA.

Then add 20  $\mu$ l of diluted ExtrAvidin-Alkaline Phosphotase (1:5,000 dilution) to each well and incubated at 37°C for 30 min. Wells are washed X3 with PBS(+Ca,Mg)+0.5% BSA. 1 tablet of p-Nitrophenol Phosphate pNPP is dissolved in 5 ml of glycine buffer (pH 10.4). 100  $\mu$ l of pNPP substrate

in glycine buffer is added to each test well. Standard wells in triplicate are prepared from the working dilution of the ExtrAvidin-Alkaline Phosphotase in glycine buffer: 1:5,000 ( $10^{0}$ ) >  $10^{-0.5}$  >  $10^{-1}$  >  $10^{-1.5}$ . 5  $\mu$ l of each dilution is added to triplicate wells and the resulting AP content in each well is 5.50 ng, 1.74 ng, 0.55 ng, 0.18 ng. 100  $\mu$ l of pNNP reagent must then be added to each of the standard wells. The plate must be incubated at 37°C for 4h. A volume of 50  $\mu$ l of 3M NaOH is added to all wells. The results are quantified on a plate reader at 405 nm. The background subtraction option is used on blank wells filled with glycine buffer only. The template is set up to indicate the concentration of AP-conjugate in each standard well [5.50 ng; 1.74 ng; 0.55 ng; 0.18 ng]. Results are indicated as amount of bound AP-conjugate in each sample.

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The studies described in this example tested activity of agonists or antagonists of the invention. However, one skilled in the art could easily modify the exemplified studies to test the activity of polynucleotides or polypeptides of the invention (e.g., gene therapy).

# Example 27: Production Of Polypeptide of the Invention For High-Throughput Screening Assays

The following protocol produces a supernatant containing polypeptide of the present invention to be tested. This supernatant can then be used in the Screening Assays described in Examples 29-37.

First, dilute Poly-D-Lysine (644 587 Boehringer-Mannheim) stock solution (1mg/ml in PBS) 1:20 in PBS (w/o calcium or magnesium 17-516F Biowhittaker) for a working solution of 50ug/ml. Add 200 ul of this solution to each well (24 well plates) and incubate at RT for 20 minutes. Be sure to distribute the solution over each well (note: a 12-channel pipetter may be used with tips on every other channel). Aspirate off the Poly-D-Lysine solution and rinse with 1ml PBS (Phosphate Buffered Saline). The PBS should remain in the well until just prior to plating the cells and plates may be poly-lysine coated in advance for up to two weeks.

Plate 293T cells (do not carry cells past P+20) at 2 x 10⁵ cells/well in .5ml DMEM(Dulbecco's Modified Eagle Medium)(with 4.5 G/L glucose and L-glutamine (12-604F Biowhittaker))/10% heat inactivated FBS(14-503F Biowhittaker)/1x Penstrep(17-602E Biowhittaker). Let the cells grow overnight.

The next day, mix together in a sterile solution basin: 300 ul Lipofectamine (18324-012 Gibco/BRL) and 5ml Optimem I (31985070 Gibco/BRL)/96-well plate. With a small volume multi-channel pipetter, aliquot approximately 2ug of an expression vector containing a polynucleotide insert, produced by the methods described in Examples 8-10, into an appropriately labeled 96-well round bottom plate. With a multi-channel pipetter, add 50ul of the Lipofectamine/Optimem I mixture to each well. Pipette up and down gently to mix. Incubate at RT 15-45 minutes. After about 20 minutes, use a multi-channel pipetter to add 150ul Optimem I

to each well. As a control, one plate of vector DNA lacking an insert should be transfected with each set of transfections.

Preferably, the transfection should be performed by tag-teaming the following tasks. By tag-teaming, hands on time is cut in half, and the cells do not spend too much time on PBS. First, person A aspirates off the media from four 24-well plates of cells, and then person B rinses each well with .5-1ml PBS. Person A then aspirates off PBS rinse, and person B, using a12-channel pipetter with tips on every other channel, adds the 200ul of DNA/Lipofectamine/Optimem I complex to the odd wells first, then to the even wells, to each row on the 24-well plates. Incubate at 37 degree C for 6 hours.

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While cells are incubating, prepare appropriate media, either 1%BSA in DMEM with 1x penstrep, or HGS CHO-5 media (116.6 mg/L of CaCl2 (anhyd); 0.00130 mg/L CuSO₄-5H₂O; 0.050 mg/L of Fe(NO₃)₃-9H₂O; 0.417 mg/L of FeSO₄-7H₂O; 311.80 mg/L of Kcl; 28.64 mg/L of MgCl2; 48.84 mg/L of MgSO4; 6995.50 mg/L of NaCl; 2400.0 mg/L of NaHCO3; 62.50 mg/L of NaH₂PO₄-H₂O; 71.02 mg/L of Na₂HPO₄; .4320 mg/L of ZnSO₄-7H₂O; .002 mg/L of Arachidonic Acid; 1.022 mg/L of Cholesterol; .070 mg/L of DL-alpha-Tocopherol-Acetate; 0.0520 mg/L of Linoleic Acid; 0.010 mg/L of Linolenic Acid; 0.010 mg/L of Myristic Acid; 0.010 mg/L of Oleic Acid; 0.010 mg/L of Palmitric Acid; 0.010 mg/L of Palmitic Acid; 100 mg/L of Pluronic F-68; 0.010 mg/L of Stearic Acid; 2.20 mg/L of Tween 80; 4551 mg/L of D-Glucose; 130.85 mg/ml of L- Alanine; 147.50 mg/ml of L-Arginine-HCL; 7.50 mg/ml of L-Asparagine-H₂0; 6.65 mg/ml of L-Aspartic Acid; 29.56 mg/ml of L-Cystine-2HCL-H₂0; 31.29 mg/ml of L-Cystine-2HCL; 7.35 mg/ml of L-Glutamic Acid; 365.0 mg/ml of L-Glutamine; 18.75 mg/ml of Glycine; 52.48 mg/ml of L-Histidine-HCL-H₂0; 106.97 mg/ml of L-Isoleucine; 111.45 mg/ml of L-Leucine: 163.75 mg/ml of L-Lysine HCL; 32.34 mg/ml of L-Methionine; 68.48 mg/ml of L-Phenylalainine; 40.0 mg/ml of L-Proline; 26.25 mg/ml of L-Serine; 101.05 mg/ml of L-Threonine; 19.22 mg/ml of L-Tryptophan; 91.79 mg/ml of L-Tryrosine-2Na-2H₂O; and 99.65 mg/ml of L-Valine; 0.0035 mg/L of Biotin; 3.24 mg/L of D-Ca Pantothenate; 11.78 mg/L of Choline Chloride; 4.65 mg/L of Folic Acid: 15.60 mg/L of i-Inositol; 3.02 mg/L of Niacinamide; 3.00 mg/L of Pyridoxal HCL; 0.031 mg/L of Pyridoxine HCL; 0.319 mg/L of Riboflavin; 3.17 mg/L of Thiamine HCL; 0.365 mg/L of Thymidine; 0.680 mg/L of Vitamin B₁₂; 25 mM of HEPES Buffer; 2.39 mg/L of Na Hypoxanthine; 0.105 mg/L of Lipoic Acid; 0.081 mg/L of Sodium Putrescine-2HCL; 55.0 mg/L of Sodium Pyruvate; 0.0067 mg/L of Sodium Selenite; 20uM of Ethanolamine; 0.122 mg/L of Ferric Citrate; 41.70 mg/L of Methyl-B-Cyclodextrin complexed with Linoleic Acid; 33.33 mg/L of Methyl-B-Cyclodextrin complexed with Oleic Acid; 10 mg/L of Methyl-B-Cyclodextrin complexed with Retinal Acetate. Adjust osmolarity to 327 mOsm) with 2mm glutamine and 1x penstrep. (BSA (81-068-3 Bayer) 100gm dissolved in 1L DMEM for a

10% BSA stock solution). Filter the media and collect 50 ul for endotoxin assay in 15ml polystyrene conical.

The transfection reaction is terminated, preferably by tag-teaming, at the end of the incubation period. Person A aspirates off the transfection media, while person B adds 1.5ml appropriate media to each well. Incubate at 37 degree C for 45 or 72 hours depending on the media used: 1%BSA for 45 hours or CHO-5 for 72 hours.

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On day four, using a 300ul multichannel pipetter, aliquot 600ul in one 1ml deep well plate and the remaining supernatant into a 2ml deep well. The supernatants from each well can then be used in the assays described in Examples 29-35.

It is specifically understood that when activity is obtained in any of the assays described below using a supernatant, the activity originates from either the polypeptide of the present invention directly (e.g., as a secreted protein) or by polypeptide of the present invention inducing expression of other proteins, which are then secreted into the supernatant. Thus, the invention further provides a method of identifying the protein in the supernatant characterized by an activity in a particular assay.

#### Example 28: Construction of GAS Reporter Construct

One signal transduction pathway involved in the differentiation and proliferation of cells is called the Jaks-STATs pathway. Activated proteins in the Jaks-STATs pathway bind to gamma activation site "GAS" elements or interferon-sensitive responsive element ("ISRE"), located in the promoter of many genes. The binding of a protein to these elements alter the expression of the associated gene.

GAS and ISRE elements are recognized by a class of transcription factors called Signal Transducers and Activators of Transcription, or "STATs." There are six members of the STATs family. Stat1 and Stat3 are present in many cell types, as is Stat2 (as response to IFN-alpha is widespread). Stat4 is more restricted and is not in many cell types though it has been found in T helper class I, cells after treatment with IL-12. Stat5 was originally called mammary growth factor, but has been found at higher concentrations in other cells including myeloid cells. It can be activated in tissue culture cells by many cytokines.

The STATs are activated to translocate from the cytoplasm to the nucleus upon tyrosine phosphorylation by a set of kinases known as the Janus Kinase ("Jaks") family. Jaks represent a distinct family of soluble tyrosine kinases and include Tyk2, Jak1, Jak2, and Jak3. These kinases display significant sequence similarity and are generally catalytically inactive in resting cells.

The Jaks are activated by a wide range of receptors summarized in the Table below. (Adapted from review by Schidler and Darnell, Ann. Rev. Biochem. 64:621-51 (1995)). A cytokine receptor family, capable of activating Jaks, is divided into two groups: (a) Class 1 includes receptors for IL-2, IL-3, IL-4, IL-6, IL-7, IL-9, IL-11, IL-12, IL-15, Epo, PRL, GH, G-

CSF, GM-CSF, LIF, CNTF, and thrombopoietin; and (b) Class 2 includes IFN-a, IFN-g, and IL-10. The Class 1 receptors share a conserved cysteine motif (a set of four conserved cysteines and one tryptophan) and a WSXWS motif (a membrane proximal region encoding Trp-Ser-Xaa-Trp-Ser (SEQ ID NO: 2)).

Thus, on binding of a ligand to a receptor, Jaks are activated, which in turn activate STATs, which then translocate and bind to GAS elements. This entire process is encompassed in the Jaks-STATs signal transduction pathway. Therefore, activation of the Jaks-STATs pathway, reflected by the binding of the GAS or the ISRE element, can be used to indicate proteins involved in the proliferation and differentiation of cells. For example, growth factors and cytokines are known to activate the Jaks-STATs pathway (See Table below). Thus, by using GAS elements linked to reporter molecules, activators of the Jaks-STATs pathway can be identified.

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<u>Ligand</u>	tyk2	<u>JAKs</u> <u>Jak1</u>	Jak2	Jak3	<u>STATS</u>	GAS(elements) or ISRE
TTN formally				•		
IFN family					122	ISRE
IFN-a/B	+	+	-	-	1,2,3	
IFN-g		+	+	-	1	GAS (IRF1>Lys6>IFP)
II-10	+	?	?	-	1,3	
gp130 family						
IL-6 (Pleiotropic)	+	+	+	?	1,3	GAS (IRF1>Lys6>IFP)
Il-11(Pleiotropic)	?	+	?	?	1,3	
OnM(Pleiotropic)	?	+	+	?	1,3	
LIF(Pleiotropic)	?	+	+	?	1,3	
CNTF(Pleiotropic)	-/+	+	+	?	1,3	
G-CSF(Pleiotropic)	?	+	?	?	1,3	
IL-12(Pleiotropic)	+		+	+	1,3	
IL-12(i lelotropie)	Ψ	-	т	T	1,5	
g-C family						
IL-2 (lymphocytes)	-	+	-	+	1,3,5	GAS
IL-4 (lymph/myeloid)	-	+	-	+	6	GAS (IRF1 = IFP >> Ly6)(IgH)
IL-7 (lymphocytes)	-	+	-	+	5	GAS
IL-9 (lymphocytes)	-	+	-	+	5	GAS
IL-13 (lymphocyte)	-	+	?	?	6	GAS
IL-15	?	+	?	+	5	GAS
140 family						
gp140 family					5	GAS (IRF1>IFP>>Ly6)
IL-3 (myeloid)	-	-	+	-	5	GAS (IRC 1211122Lyo)
IL-5 (myeloid)	-	-	+	-	5	GAS
GM-CSF (myeloid)	-	-	+	-	3	GAS
Growth hormone family						
GH	?	_	+	-	5	
PRL	?	+/-	+	-	1,3,5	
EPO	?	_	+	-	5	GAS(B-
CAS>IRF1=IFP>>Ly6	)					•
Receptor Tyrosine Kinases						
EGF	?	+	+	-	1,3	GAS (IRF1)
PDGF	?	+	+	-	1,3	
CSF-1	?	+	+	-	1,3	GAS (not IRF1)

To construct a synthetic GAS containing promoter element, which is used in the Biological Assays described in Examples 29-30, a PCR based strategy is employed to generate a GAS-SV40 promoter sequence. The 5' primer contains four tandem copies of the GAS binding site found in the IRF1 promoter and previously demonstrated to bind STATs upon induction with a range of cytokines (Rothman et al., Immunity 1:457-468 (1994).), although other GAS or ISRE elements can be used instead. The 5' primer also contains 18bp of sequence complementary to the SV40 early promoter sequence and is flanked with an XhoI site. The sequence of the 5' primer is:

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5':GCGCCTCGAGATTTCCCCGAAATCTAGATTTCCCCGAAATGATTTCCCCGAA ATGATTTCCCCGAAATATCTGCCATCTCAATTAG:3' (SEQ ID NO: 3)

The downstream primer is complementary to the SV40 promoter and is flanked with a Hind III site: 5':GCGGCAAGCTTTTTGCAAAGCCTAGGC:3' (SEQ ID NO: 4)

PCR amplification is performed using the SV40 promoter template present in the B-gal:promoter plasmid obtained from Clontech. The resulting PCR fragment is digested with XhoI/Hind III and subcloned into BLSK2-. (Stratagene.) Sequencing with forward and reverse primers confirms that the insert contains the following sequence:

With this GAS promoter element linked to the SV40 promoter, a GAS:SEAP2 reporter construct is next engineered. Here, the reporter molecule is a secreted alkaline phosphatase, or "SEAP." Clearly, however, any reporter molecule can be instead of SEAP, in this or in any of the other Examples. Well known reporter molecules that can be used instead of SEAP include chloramphenical acetyltransferase (CAT), luciferase, alkaline phosphatase, B-galactosidase, green fluorescent protein (GFP), or any protein detectable by an antibody.

The above sequence confirmed synthetic GAS-SV40 promoter element is subcloned into the pSEAP-Promoter vector obtained from Clontech using HindIII and XhoI, effectively replacing the SV40 promoter with the amplified GAS:SV40 promoter element, to create the GAS-SEAP vector. However, this vector does not contain a neomycin resistance gene, and therefore, is not preferred for mammalian expression systems.

Thus, in order to generate mammalian stable cell lines expressing the GAS-SEAP reporter, the GAS-SEAP cassette is removed from the GAS-SEAP vector using SalI and NotI, and inserted into a backbone vector containing the neomycin resistance gene, such as pGFP-1 (Clontech), using these restriction sites in the multiple cloning site, to create the GAS-SEAP/Neo vector. Once this

vector is transfected into mammalian cells, this vector can then be used as a reporter molecule for GAS binding as described in Examples 29-30.

Other constructs can be made using the above description and replacing GAS with a different promoter sequence. For example, construction of reporter molecules containing EGR and NF-KB promoter sequences are described in Example 31. However, many other promoters can be substituted using the protocols described in these Examples. For instance, SRE, IL-2, NFAT, or Osteocalcin promoters can be substituted, alone or in combination (e.g., GAS/NF-KB/EGR, GAS/NF-KB, Il-2/NFAT, or NF-KB/GAS). Similarly, other cell lines can be used to test reporter construct activity, such as HELA (epithelial), HUVEC (endothelial), Reh (B-cell), Saos-2 (osteoblast), HUVAC (aortic), or Cardiomyocyte.

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### Example 29: High-Throughput Screening Assay for T-cell Activity.

The following protocol is used to assess T-cell activity by identifying factors, and determining whether supernate containing a polypeptide of the invention proliferates and/or differentiates T-cells. T-cell activity is assessed using the GAS/SEAP/Neo construct produced in Example 28. Thus, factors that increase SEAP activity indicate the ability to activate the Jaks-STATS signal transduction pathway. The T-cell used in this assay is Jurkat T-cells (ATCC Accession No. TIB-152), although Molt-3 cells (ATCC Accession No. CRL-1552) and Molt-4 cells (ATCC Accession No. CRL-1582) cells can also be used.

Jurkat T-cells are lymphoblastic CD4+ Th1 helper cells. In order to generate stable cell lines, approximately 2 million Jurkat cells are transfected with the GAS-SEAP/neo vector using DMRIE-C (Life Technologies)(transfection procedure described below). The transfected cells are seeded to a density of approximately 20,000 cells per well and transfectants resistant to 1 mg/ml genticin selected. Resistant colonies are expanded and then tested for their response to increasing concentrations of interferon gamma. The dose response of a selected clone is demonstrated.

Specifically, the following protocol will yield sufficient cells for 75 wells containing 200 ul of cells. Thus, it is either scaled up, or performed in multiple to generate sufficient cells for multiple 96 well plates. Jurkat cells are maintained in RPMI + 10% serum with 1%Pen-Strep. Combine 2.5 mls of OPTI-MEM (Life Technologies) with 10 ug of plasmid DNA in a T25 flask. Add 2.5 ml OPTI-MEM containing 50 ul of DMRIE-C and incubate at room temperature for 15-45 mins.

During the incubation period, count cell concentration, spin down the required number of cells (10⁷ per transfection), and resuspend in OPTI-MEM to a final concentration of 10⁷ cells/ml. Then add 1ml of 1 x 10⁷ cells in OPTI-MEM to T25 flask and incubate at 37 degree C for 6 hrs. After the incubation, add 10 ml of RPMI + 15% serum.

The Jurkat:GAS-SEAP stable reporter lines are maintained in RPMI + 10% serum, 1 mg/ml Genticin, and 1% Pen-Strep. These cells are treated with supernatants containing polypeptide of the present invention or polypeptide of the present invention induced polypeptides as produced by the protocol described in Example 27.

On the day of treatment with the supernatant, the cells should be washed and resuspended in fresh RPMI + 10% serum to a density of 500,000 cells per ml. The exact number of cells required will depend on the number of supernatants being screened. For one 96 well plate, approximately 10 million cells (for 10 plates, 100 million cells) are required.

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Transfer the cells to a triangular reservoir boat, in order to dispense the cells into a 96 well dish, using a 12 channel pipette. Using a 12 channel pipette, transfer 200 ul of cells into each well (therefore adding 100, 000 cells per well).

After all the plates have been seeded, 50 ul of the supernatants are transferred directly from the 96 well plate containing the supernatants into each well using a 12 channel pipette. In addition, a dose of exogenous interferon gamma (0.1, 1.0, 10 ng) is added to wells H9, H10, and H11 to serve as additional positive controls for the assay.

The 96 well dishes containing Jurkat cells treated with supernatants are placed in an incubator for 48 hrs (note: this time is variable between 48-72 hrs). 35 ul samples from each well are then transferred to an opaque 96 well plate using a 12 channel pipette. The opaque plates should be covered (using sellophene covers) and stored at -20 degree C until SEAP assays are performed according to Example 32. The plates containing the remaining treated cells are placed at 4 degree C and serve as a source of material for repeating the assay on a specific well if desired.

As a positive control, 100 Unit/ml interferon gamma can be used which is known to activate Jurkat T cells. Over 30 fold induction is typically observed in the positive control wells.

The above protocol may be used in the generation of both transient, as well as, stable transfected cells, which would be apparent to those of skill in the art.

### Example 30: High-Throughput Screening Assay Identifying Myeloid Activity

The following protocol is used to assess myeloid activity of polypeptide of the present invention by determining whether polypeptide of the present invention proliferates and/or differentiates myeloid cells. Myeloid cell activity is assessed using the GAS/SEAP/Neo construct produced in Example 28. Thus, factors that increase SEAP activity indicate the ability to activate the Jaks-STATS signal transduction pathway. The myeloid cell used in this assay is U937, a premonocyte cell line, although TF-1, HL60, or KG1 can be used.

To transiently transfect U937 cells with the GAS/SEAP/Neo construct produced in Example 28, a DEAE-Dextran method (Kharbanda et. al., 1994, Cell Growth & Differentiation,

5:259-265) is used. First, harvest 2x10⁷ U937 cells and wash with PBS. The U937 cells are usually grown in RPMI 1640 medium containing 10% heat-inactivated fetal bovine serum (FBS) supplemented with 100 units/ml penicillin and 100 mg/ml streptomycin.

Next, suspend the cells in 1 ml of 20 mM Tris-HCl (pH 7.4) buffer containing 0.5 mg/ml DEAE-Dextran, 8 ug GAS-SEAP2 plasmid DNA, 140 mM NaCl, 5 mM KCl, 375 uM Na₂HPO₄.7H₂O, 1 mM MgCl₂, and 675 uM CaCl₂. Incubate at 37 degrees C for 45 min.

Wash the cells with RPMI 1640 medium containing 10% FBS and then resuspend in 10 ml complete medium and incubate at 37 degree C for 36 hr.

The GAS-SEAP/U937 stable cells are obtained by growing the cells in 400 ug/ml G418. The G418-free medium is used for routine growth but every one to two months, the cells should be re-grown in 400 ug/ml G418 for couple of passages.

These cells are tested by harvesting  $1x10^8$  cells (this is enough for ten 96-well plates assay) and wash with PBS. Suspend the cells in 200 ml above described growth medium, with a final density of  $5x10^5$  cells/ml. Plate 200 ul cells per well in the 96-well plate (or  $1x10^5$  cells/well).

Add 50 ul of the supernatant prepared by the protocol described in Example 27. Incubate at 37 degee C for 48 to 72 hr. As a positive control, 100 Unit/ml interferon gamma can be used which is known to activate U937 cells. Over 30 fold induction is typically observed in the positive control wells. SEAP assay the supernatant according to the protocol described in Example 32.

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# Example 31: High-Throughput Screening Assay for T-cell Activity

NF-KB (Nuclear Factor KB) is a transcription factor activated by a wide variety of agents including the inflammatory cytokines IL-1 and TNF, CD30 and CD40, lymphotoxin-alpha and lymphotoxin-beta, by exposure to LPS or thrombin, and by expression of certain viral gene products. As a transcription factor, NF-KB regulates the expression of genes involved in immune cell activation, control of apoptosis (NF- KB appears to shield cells from apoptosis), B and T-cell development, anti-viral and antimicrobial responses, and multiple stress responses.

In non-stimulated conditions, NF- KB is retained in the cytoplasm with I-KB (Inhibitor KB). However, upon stimulation, I- KB is phosphorylated and degraded, causing NF- KB to shuttle to the nucleus, thereby activating transcription of target genes. Target genes activated by NF- KB include IL-2, IL-6, GM-CSF, ICAM-1 and class 1 MHC.

Due to its central role and ability to respond to a range of stimuli, reporter constructs utilizing the NF-KB promoter element are used to screen the supernatants produced in Example 27. Activators or inhibitors of NF-KB would be useful in detecting, preventing, diagnosing, prognosticating, treating, and/or ameliorating diseases. For example, inhibitors of NF-KB could

be used to treat those diseases related to the acute or chronic activation of NF-KB, such as rheumatoid arthritis.

To construct a vector containing the NF-KB promoter element, a PCR based strategy is employed. The upstream primer contains four tandem copies of the NF-KB binding site (GGGGACTTTCCC) (SEQ ID NO: 8), 18 bp of sequence complementary to the 5' end of the SV40 early promoter sequence, and is flanked with an XhoI site:

5':GCGGCCTCGAGGGGACTTTCCCGGGGACTTTCCGGGGACTTT CCATCCTGCCATCTCAATTAG:3' (SEQ ID NO: 9)

The downstream primer is complementary to the 3' end of the SV40 promoter and is flanked with a Hind III site:

5':GCGGCAAGCTTTTTGCAAAGCCTAGGC:3' (SEQ ID NO: 4)

PCR amplification is performed using the SV40 promoter template present in the pB-gal:promoter plasmid obtained from Clontech. The resulting PCR fragment is digested with XhoI and Hind III and subcloned into BLSK2-. (Stratagene) Sequencing with the T7 and T3 primers confirms the insert contains the following sequence:

Next, replace the SV40 minimal promoter element present in the pSEAP2-promoter plasmid (Clontech) with this NF-KB/SV40 fragment using XhoI and HindIII. However, this vector does not contain a neomycin resistance gene, and therefore, is not preferred for mammalian expression systems.

In order to generate stable mammalian cell lines, the NF-KB/SV40/SEAP cassette is removed from the above NF-KB/SEAP vector using restriction enzymes SalI and NotI, and inserted into a vector containing neomycin resistance. Particularly, the NF-KB/SV40/SEAP cassette was inserted into pGFP-1 (Clontech), replacing the GFP gene, after restricting pGFP-1 with SalI and NotI.

Once NF-KB/SV40/SEAP/Neo vector is created, stable Jurkat T-cells are created and maintained according to the protocol described in Example 29. Similarly, the method for assaying supernatants with these stable Jurkat T-cells is also described in Example 29. As a positive control, exogenous TNF alpha (0.1,1, 10 ng) is added to wells H9, H10, and H11, with a 5-10 fold activation typically observed.

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#### Example 32: Assay for SEAP Activity

As a reporter molecule for the assays described in Examples 29-31, SEAP activity is assayed using the Tropix Phospho-light Kit (Cat. BP-400) according to the following general procedure. The Tropix Phospho-light Kit supplies the Dilution, Assay, and Reaction Buffers used below.

Prime a dispenser with the 2.5x Dilution Buffer and dispense 15 ul of 2.5x dilution buffer into Optiplates containing 35 ul of a supernatant. Seal the plates with a plastic sealer and incubate at 65 degree C for 30 min. Separate the Optiplates to avoid uneven heating.

Cool the samples to room temperature for 15 minutes. Empty the dispenser and prime with the Assay Buffer. Add 50 ml Assay Buffer and incubate at room temperature 5 min. Empty the dispenser and prime with the Reaction Buffer (see the Table below). Add 50 ul Reaction Buffer and incubate at room temperature for 20 minutes. Since the intensity of the chemiluminescent signal is time dependent, and it takes about 10 minutes to read 5 plates on a luminometer, thus one should treat 5 plates at each time and start the second set 10 minutes later.

Read the relative light unit in the luminometer. Set H12 as blank, and print the results. An increase in chemiluminescence indicates reporter activity.

Reaction Buffer Formulation:

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# of plates	Rxn buffer diluent (ml)	CSPD (ml)
10	60	3
11	65	3.25
12	70	3.5
13	75	3.75
14	80	4
15	85	4.25
16	90	4.5
17	95	4.75
18	100	5
19	105	5.25
20	110	5.5
21	115	5.75
22	120	6
23	125	6.25
24	130	6.5
25	135	6.75
26	140	7
27	145	7.25
28	150	7.5
29	155	7.75
30	160	8
31	165	8.25
32	170	8.5
33	175	8.75
34	180	9 .

# of plates	Rxn buffer diluent (ml)	CSPD (ml)
35	185	9.25
36	190	9.5
37	195	9.75
38	200	10
39	205	10.25
40	210	10.5
41	215	10.75
42	220	11
43	225	11.25
44	230	11.5
45	235	11.75
46	240	12
47	245	12.25
48	250	12.5
49	255	12.75
50	260	13

Example 33: High-Throughput Screening Assay Identifying Changes in Small Molecule

Concentration and Membrane Permeability

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Binding of a ligand to a receptor is known to alter intracellular levels of small molecules, such as calcium, potassium, sodium, and pH, as well as alter membrane potential. These alterations can be measured in an assay to identify supernatants which bind to receptors of a particular cell. Although the following protocol describes an assay for calcium, this protocol can easily be modified to detect changes in potassium, sodium, pH, membrane potential, or any other small molecule which is detectable by a fluorescent probe.

The following assay uses Fluorometric Imaging Plate Reader ("FLIPR") to measure changes in fluorescent molecules (Molecular Probes) that bind small molecules. Clearly, any fluorescent molecule detecting a small molecule can be used instead of the calcium fluorescent molecule, fluo-4 (Molecular Probes, Inc.; catalog no. F-14202), used here.

For adherent cells, seed the cells at 10,000 -20,000 cells/well in a Co-star black 96-well plate with clear bottom. The plate is incubated in a CO₂ incubator for 20 hours. The adherent cells are washed two times in Biotek washer with 200 ul of HBSS (Hank's Balanced Salt Solution) leaving 100 ul of buffer after the final wash.

A stock solution of 1 mg/ml fluo-4 is made in 10% pluronic acid DMSO. To load the cells with fluo-4, 50 ul of 12 ug/ml fluo-4 is added to each well. The plate is incubated at 37 degrees C in a CO₂ incubator for 60 min. The plate is washed four times in the Biotek washer with HBSS leaving 100 ul of buffer.

For non-adherent cells, the cells are spun down from culture media. Cells are resuspended to 2-5x10⁶ cells/ml with HBSS in a 50-ml conical tube. 4 ul of 1 mg/ml fluo-4 solution

in 10% pluronic acid DMSO is added to each ml of cell suspension. The tube is then placed in a 37 degrees C water bath for 30-60 min. The cells are washed twice with HBSS, resuspended to  $1 \times 10^6$  cells/ml, and dispensed into a microplate, 100 ul/well. The plate is centrifuged at 1000 rpm for 5 min. The plate is then washed once in Denley Cell Wash with 200 ul, followed by an aspiration step to 100 ul final volume.

For a non-cell based assay, each well contains a fluorescent molecule, such as fluo-4. The supernatant is added to the well, and a change in fluorescence is detected.

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To measure the fluorescence of intracellular calcium, the FLIPR is set for the following parameters: (1) System gain is 300-800 mW; (2) Exposure time is 0.4 second; (3) Camera F/stop is F/2; (4) Excitation is 488 nm; (5) Emission is 530 nm; and (6) Sample addition is 50 ul. Increased emission at 530 nm indicates an extracellular signaling event caused by the a molecule, either polypeptide of the present invention or a molecule induced by polypeptide of the present invention, which has resulted in an increase in the intracellular Ca⁺⁺ concentration.

## Example 34: High-Throughput Screening Assay Identifying Tyrosine Kinase Activity

The Protein Tyrosine Kinases (PTK) represent a diverse group of transmembrane and cytoplasmic kinases. Within the Receptor Protein Tyrosine Kinase RPTK) group are receptors for a range of mitogenic and metabolic growth factors including the PDGF, FGF, EGF, NGF, HGF and Insulin receptor subfamilies. In addition there are a large family of RPTKs for which the corresponding ligand is unknown. Ligands for RPTKs include mainly secreted small proteins, but also membrane-bound and extracellular matrix proteins.

Activation of RPTK by ligands involves ligand-mediated receptor dimerization, resulting in transphosphorylation of the receptor subunits and activation of the cytoplasmic tyrosine kinases. The cytoplasmic tyrosine kinases include receptor associated tyrosine kinases of the src-family (e.g., src, yes, lck, lyn, fyn) and non-receptor linked and cytosolic protein tyrosine kinases, such as the Jak family, members of which mediate signal transduction triggered by the cytokine superfamily of receptors (e.g., the Interleukins, Interferons, GM-CSF, and Leptin).

Because of the wide range of known factors capable of stimulating tyrosine kinase activity, identifying whether polypeptide of the present invention or a molecule induced by polypeptide of the present invention is capable of activating tyrosine kinase signal transduction pathways is of interest. Therefore, the following protocol is designed to identify such molecules capable of activating the tyrosine kinase signal transduction pathways.

Seed target cells (e.g., primary keratinocytes) at a density of approximately 25,000 cells per well in a 96 well Loprodyne Silent Screen Plates purchased from Nalge Nunc (Naperville, IL). The plates are sterilized with two 30 minute rinses with 100% ethanol, rinsed with water and dried

overnight. Some plates are coated for 2 hr with 100 ml of cell culture grade type I collagen (50 mg/ml), gelatin (2%) or polylysine (50 mg/ml), all of which can be purchased from Sigma Chemicals (St. Louis, MO) or 10% Matrigel purchased from Becton Dickinson (Bedford,MA), or calf serum, rinsed with PBS and stored at 4 degree C. Cell growth on these plates is assayed by seeding 5,000 cells/well in growth medium and indirect quantitation of cell number through use of alamarBlue as described by the manufacturer Alamar Biosciences, Inc. (Sacramento, CA) after 48 hr. Falcon plate covers #3071 from Becton Dickinson (Bedford,MA) are used to cover the Loprodyne Silent Screen Plates. Falcon Microtest III cell culture plates can also be used in some proliferation experiments.

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To prepare extracts, A431 cells are seeded onto the nylon membranes of Loprodyne plates (20,000/200ml/well) and cultured overnight in complete medium. Cells are quiesced by incubation in serum-free basal medium for 24 hr. After 5-20 minutes treatment with EGF (60ng/ml) or 50 ul of the supernatant produced in Example 27, the medium was removed and 100 ml of extraction buffer ((20 mM HEPES pH 7.5, 0.15 M NaCl, 1% Triton X-100, 0.1% SDS, 2 mM Na3VO4, 2 mM Na4P2O7 and a cocktail of protease inhibitors (# 1836170) obtained from Boeheringer Mannheim (Indianapolis, IN)) is added to each well and the plate is shaken on a rotating shaker for 5 minutes at 4°C. The plate is then placed in a vacuum transfer manifold and the extract filtered through the 0.45 mm membrane bottoms of each well using house vacuum. Extracts are collected in a 96-well catch/assay plate in the bottom of the vacuum manifold and immediately placed on ice. To obtain extracts clarified by centrifugation, the content of each well, after detergent solubilization for 5 minutes, is removed and centrifuged for 15 minutes at 4 degree C at 16,000 x g.

Test the filtered extracts for levels of tyrosine kinase activity. Although many methods of detecting tyrosine kinase activity are known, one method is described here.

Generally, the tyrosine kinase activity of a supernatant is evaluated by determining its ability to phosphorylate a tyrosine residue on a specific substrate (a biotinylated peptide). Biotinylated peptides that can be used for this purpose include PSK1 (corresponding to amino acids 6-20 of the cell division kinase cdc2-p34) and PSK2 (corresponding to amino acids 1-17 of gastrin). Both peptides are substrates for a range of tyrosine kinases and are available from Boehringer Mannheim.

The tyrosine kinase reaction is set up by adding the following components in order. First, add 10ul of 5uM Biotinylated Peptide, then 10ul ATP/Mg₂₊ (5mM ATP/50mM MgCl₂), then 10ul of 5x Assay Buffer (40mM imidazole hydrochloride, pH7.3, 40 mM beta-glycerophosphate, 1mM EGTA, 100mM MgCl₂, 5 mM MnCl₂, 0.5 mg/ml BSA), then 5ul of Sodium Vanadate(1mM), and then 5ul of water. Mix the components gently and preincubate the reaction

mix at 30 degree C for 2 min. Initial the reaction by adding 10ul of the control enzyme or the filtered supernatant.

The tyrosine kinase assay reaction is then terminated by adding 10 ul of 120mm EDTA and place the reactions on ice.

Tyrosine kinase activity is determined by transferring 50 ul aliquot of reaction mixture to a microtiter plate (MTP) module and incubating at 37 degree C for 20 min. This allows the streptavidin coated 96 well plate to associate with the biotinylated peptide. Wash the MTP module with 300ul/well of PBS four times. Next add 75 ul of anti-phospotyrosine antibody conjugated to horse radish peroxidase(anti-P-Tyr-POD(0.5u/ml)) to each well and incubate at 37 degree C for one hour. Wash the well as above.

Next add 100ul of peroxidase substrate solution (Boehringer Mannheim) and incubate at room temperature for at least 5 mins (up to 30 min). Measure the absorbance of the sample at 405 nm by using ELISA reader. The level of bound peroxidase activity is quantitated using an ELISA reader and reflects the level of tyrosine kinase activity.

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## Example 35: High-Throughput Screening Assay Identifying Phosphorylation Activity

As a potential alternative and/or complement to the assay of protein tyrosine kinase activity described in Example 34, an assay which detects activation (phosphorylation) of major intracellular signal transduction intermediates can also be used. For example, as described below one particular assay can detect tyrosine phosphorylation of the Erk-1 and Erk-2 kinases. However, phosphorylation of other molecules, such as Raf, JNK, p38 MAP, Map kinase kinase (MEK), MEK kinase, Src, Muscle specific kinase (MuSK), IRAK, Tec, and Janus, as well as any other phosphoserine, phosphotyrosine, or phosphothreonine molecule, can be detected by substituting these molecules for Erk-1 or Erk-2 in the following assay.

Specifically, assay plates are made by coating the wells of a 96-well ELISA plate with 0.1ml of protein G (1ug/ml) for 2 hr at room temp, (RT). The plates are then rinsed with PBS and blocked with 3% BSA/PBS for 1 hr at RT. The protein G plates are then treated with 2 commercial monoclonal antibodies (100ng/well) against Erk-1 and Erk-2 (1 hr at RT) (Santa Cruz Biotechnology). (To detect other molecules, this step can easily be modified by substituting a monoclonal antibody detecting any of the above described molecules.) After 3-5 rinses with PBS,

the plates are stored at 4-degree C until use.

A431 cells are seeded at 20,000/well in a 96-well Loprodyne filterplate and cultured overnight in growth medium. The cells are then starved for 48 hr in basal medium (DMEM) and then treated with EGF (6ng/well) or 50 ul of the supernatants obtained in Example 27 for 5-20 minutes. The cells are then solubilized and extracts filtered directly into the assay plate.

After incubation with the extract for 1 hr at RT, the wells are again rinsed. As a positive control, a commercial preparation of MAP kinase (10ng/well) is used in place of A431 extract. Plates are then treated with a commercial polyclonal (rabbit) antibody (1ug/ml) which specifically recognizes the phosphorylated epitope of the Erk-1 and Erk-2 kinases (1 hr at RT). This antibody is biotinylated by standard procedures. The bound polyclonal antibody is then quantitated by successive incubations with Europium-streptavidin and Europium fluorescence enhancing reagent in the Wallac DELFIA instrument (time-resolved fluorescence). An increased fluorescent signal over background indicates a phosphorylation by polypeptide of the present invention or a molecule induced by polypeptide of the present invention.

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# Example 36: Assay for the Stimulation of Bone Marrow CD34+ Cell Proliferation

This assay is based on the ability of human CD34+ to proliferate in the presence of hematopoietic growth factors and evaluates the ability of isolated polypeptides expressed in mammalian cells to stimulate proliferation of CD34+ cells.

It has been previously shown that most mature precursors will respond to only a single signal. More immature precursors require at least two signals to respond. Therefore, to test the effect of polypeptides on hematopoietic activity of a wide range of progenitor cells, the assay contains a given polypeptide in the presence or absence of other hematopoietic growth factors. Isolated cells are cultured for 5 days in the presence of Stem Cell Factor (SCF) in combination with tested sample. SCF alone has a very limited effect on the proliferation of bone marrow (BM) cells, acting in such conditions only as a "survival" factor. However, combined with any factor exhibiting stimulatory effect on these cells (e.g., IL-3), SCF will cause a synergistic effect. Therefore, if the tested polypeptide has a stimulatory effect on hematopoietic progenitors, such activity can be easily detected. Since normal BM cells have a low level of cycling cells, it is likely that any inhibitory effect of a given polypeptide, or agonists or antagonists thereof, might not be detected. Accordingly, assays for an inhibitory effect on progenitors is preferably tested in cells that are first subjected to *in vitro* stimulation with SCF+IL+3, and then contacted with the compound that is being evaluated for inhibition of such induced proliferation.

Briefly, CD34+ cells are isolated using methods known in the art. The cells are thawed and resuspended in medium (QBSF 60 serum-free medium with 1% L-glutamine (500ml) Quality Biological, Inc., Gaithersburg, MD Cat# 160-204-101). After several gentle centrifugation steps at 200 x g, cells are allowed to rest for one hour. The cell count is adjusted to 2.5 x 10⁵ cells/ml. During this time, 100 µl of sterile water is added to the peripheral wells of a 96-well plate. The cytokines that can be tested with a given polypeptide in this assay is rhSCF (R&D Systems, Minneapolis, MN, Cat# 255-SC) at 50 ng/ml alone and in combination with rhSCF and rhIL-3

(R&D Systems, Minneapolis, MN, Cat# 203-ML) at 30 ng/ml. After one hour, 10  $\mu$ l of prepared cytokines, 50  $\mu$ l of the supernatants prepared in Example 27 (supernatants at 1:2 dilution = 50  $\mu$ l) and 20  $\mu$ l of diluted cells are added to the media which is already present in the wells to allow for a final total volume of 100  $\mu$ l. The plates are then placed in a 37°C/5% CO₂ incubator for five days.

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Eighteen hours before the assay is harvested, 0.5 μCi/well of [3H] Thymidine is added in a 10 μl volume to each well to determine the proliferation rate. The experiment is terminated by harvesting the cells from each 96-well plate to a filtermat using the Tomtec Harvester 96. After harvesting, the filtermats are dried, trimmed and placed into OmniFilter assemblies consisting of one OmniFilter plate and one OmniFilter Tray. 60 μl Microscint is added to each well and the plate sealed with TopSeal-A press-on sealing film A bar code 15 sticker is affixed to the first plate for counting. The sealed plates are then loaded and the level of radioactivity determined via the Packard Top Count and the printed data collected for analysis. The level of radioactivity reflects the amount of cell proliferation.

The studies described in this example test the activity of a given polypeptide to stimulate bone marrow CD34+ cell proliferation. One skilled in the art could easily modify the exemplified studies to test the activity of polynucleotides (e.g., gene therapy), antibodies, agonists, and/or antagonists and fragments and variants thereof. As a nonlimiting example, potential antagonists tested in this assay would be expected to inhibit cell proliferation in the presence of cytokines and/or to increase the inhibition of cell proliferation in the presence of cytokines and a given polypeptide. In contrast, potential agonists tested in this assay would be expected to enhance cell proliferation and/or to decrease the inhibition of cell proliferation in the presence of cytokines and a given polypeptide.

The ability of a gene to stimulate the proliferation of bone marrow CD34+ cells indicates that polynucleotides and polypeptides corresponding to the gene are useful for the detection, prevention, diagnosis, prognostication, treatment, and/or amelioration of disorders affecting the immune system and hematopoiesis. Representative uses are described in the "Immune Activity" and "Infectious Disease" sections above, and elsewhere herein.

# Example 37: Assay for Extracellular Matrix Enhanced Cell Response (EMECR)

The objective of the Extracellular Matrix Enhanced Cell Response (EMECR) assay is to identify gene products (e.g., isolated polypeptides) that act on the hematopoietic stem cells in the context of the extracellular matrix (ECM) induced signal.

Cells respond to the regulatory factors in the context of signal(s) received from the surrounding microenvironment. For example, fibroblasts, and endothelial and epithelial stem cells fail to replicate in the absence of signals from the ECM. Hematopoietic stem cells can undergo

self-renewal in the bone marrow, but not in *in vitro* suspension culture. The ability of stem cells to undergo self-renewal *in vitro* is dependent upon their interaction with the stromal cells and the ECM protein fibronectin (fn). Adhesion of cells to fn is mediated by the  $\alpha_5.\beta_1$  and  $\alpha_4.\beta_1$  integrin receptors, which are expressed by human and mouse hematopoietic stem cells. The factor(s) which integrate with the ECM environment and are responsible for stimulating stem cell self-renewal havea not yet been identified. Discovery of such factors should be of great interest in gene therapy and bone marrow transplant applications

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Briefly, polystyrene, non tissue culture treated, 96-well plates are coated with fn fragment at a coating concentration of 0.2 μg/ cm². Mouse bone marrow cells are plated (1,000 cells/well) in 0.2 ml of serum-free medium. Cells cultured in the presence of IL-3 (5 ng/ml) + SCF (50 ng/ml) would serve as the positive control, conditions under which little self-renewal but pronounced differentiation of the stem cells is to be expected. Gene products of the invention (e.g., including, but not limited to, polynucleotides and polypeptides of the present invention, and supernatants produced in Example 27), are tested with appropriate negative controls in the presence and absence of SCF(5.0 ng/ml), where test factor supernatants represent 10% of the total assay volume. The plated cells are then allowed to grow by incubating in a low oxygen environment (5% CO₂, 7% O₂, and 88% N₂) tissue culture incubator for 7 days. The number of proliferating cells within the wells is then quantitated by measuring thymidine incorporation into cellular DNA. Verification of the positive hits in the assay will require phenotypic characterization of the cells, which can be accomplished by scaling up of the culture system and using appropriate antibody reagents against cell surface antigens and FACScan.

One skilled in the art could easily modify the exemplified studies to test the activity of polynucleotides (e.g., gene therapy), antibodies, agonists, and/or antagonists and fragments and variants thereof.

If a particular polypeptide of the present invention is found to be a stimulator of hematopoietic progenitors, polynucleotides and polypeptides corresponding to the gene encoding said polypeptide may be useful for the detection, prevention, diagnosis, prognostication, treatment, and/or amelioration of disorders affecting the immune system and hematopoiesis. Representative uses are described in the "Immune Activity" and "Infectious Disease" sections above, and elsewhere herein. The gene product may also be useful in the expansion of stem cells and committed progenitors of various blood lineages, and in the differentiation and/or proliferation of various cell types.

Additionally, the polynucleotides and/or polypeptides of the gene of interest and/or agonists and/or antagonists thereof, may also be employed to inhibit the proliferation and differentiation of hematopoietic cells and therefore may be employed to protect bone marrow stem

cells from chemotherapeutic agents during chemotherapy. This antiproliferative effect may allow administration of higher doses of chemotherapeutic agents and, therefore, more effective chemotherapeutic treatment.

Moreover, polynucleotides and polypeptides corresponding to the gene of interest may also be useful for the detection, prevention, diagnosis, prognostication, treat, and/or amelioration of hematopoietic and hematologic related disorders such as, for example, anemia, pancytopenia, leukopenia, thrombocytopenia or leukemia since stromal cells are important in the production of cells of hematopoietic lineages. The uses include bone marrow cell ex-vivo culture, bone marrow transplantation, bone marrow reconstitution, radiotherapy or chemotherapy of neoplasia.

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#### Example 38: Human Dermal Fibroblast and Aortic Smooth Muscle Cell Proliferation

The polypeptide of interest is added to cultures of normal human dermal fibroblasts (NHDF) and human aortic smooth muscle cells (AoSMC) and two co-assays are performed with each sample. The first assay examines the effect of the polypeptide of interest on the proliferation of normal human dermal fibroblasts (NHDF) or aortic smooth muscle cells (AoSMC). Aberrant growth of fibroblasts or smooth muscle cells is a part of several pathological processes, including fibrosis, and restenosis. The second assay examines IL6 production by both NHDF and SMC. IL6 production is an indication of functional activation. Activated cells will have increased production of a number of cytokines and other factors, which can result in a proinflammatory or immunomodulatory outcome. Assays are run with and without co-TNFa stimulation, in order to check for costimulatory or inhibitory activity.

Briefly, on day 1, 96-well black plates are set up with 1000 cells/well (NHDF) or 2000 cells/well (AoSMC) in 100 μl culture media. NHDF culture media contains: Clonetics FB basal media, 1mg/ml hFGF, 5mg/ml insulin, 50mg/ml gentamycin, 2%FBS, while AoSMC culture media contains Clonetics SM basal media, 0.5 μg/ml hEGF, 5mg/ml insulin, 1μg/ml hFGF, 50mg/ml gentamycin, 50 μg/ml Amphotericin B, 5%FBS. After incubation at 37°C for at least 4-5 hours culture media is aspirated and replaced with growth arrest media. Growth arrest media for NHDF contains fibroblast basal media, 50mg/ml gentamycin, 2% FBS, while growth arrest media for AoSMC contains SM basal media, 50mg/ml gentamycin, 50μg/ml Amphotericin B, 0.4% FBS. Incubate at 37 °C until day 2.

On day 2, serial dilutions and templates of the polypeptide of interest are designed such that they always include media controls and known-protein controls. For both stimulation and inhibition experiments, proteins are diluted in growth arrest media. For inhibition experiments, TNFa is added to a final concentration of 2ng/ml (NHDF) or 5ng/ml (AoSMC). Add 1/3 vol

media containing controls or polypeptides of the present invention and incubate at 37 degrees C/5% CO₂ until day 5.

Transfer  $60\mu$ l from each well to another labeled 96-well plate, cover with a plate-sealer, and store at 4 degrees C until Day 6 (for IL6 ELISA). To the remaining  $100 \mu$ l in the cell culture plate, aseptically add Alamar Blue in an amount equal to 10% of the culture volume ( $10\mu$ l). Return plates to incubator for 3 to 4 hours. Then measure fluorescence with excitation at 530nm and emission at 590nm using the CytoFluor. This yields the growth stimulation/inhibition data.

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On day 5, the IL6 ELISA is performed by coating a 96 well plate with 50-100 ul/well of Anti-Human IL6 Monoclonal antibody diluted in PBS, pH 7.4, incubate ON at room temperature.

On day 6, empty the plates into the sink and blot on paper towels. Prepare Assay Buffer containing PBS with 4% BSA. Block the plates with 200  $\mu$ I/well of Pierce Super Block blocking buffer in PBS for 1-2 hr and then wash plates with wash buffer (PBS, 0.05% Tween-20). Blot plates on paper towels. Then add 50  $\mu$ I/well of diluted Anti-Human IL-6 Monoclonal, Biotinlabeled antibody at 0.50 mg/ml. Make dilutions of IL-6 stock in media (30, 10, 3, 1, 0.3, 0 ng/ml). Add duplicate samples to top row of plate. Cover the plates and incubate for 2 hours at RT on shaker.

Plates are washed with wash buffer and blotted on paper towels. Dilute EU-labeled Streptavidin 1:1000 in Assay buffer, and add 100 µl/well. Cover the plate and incubate 1 h at RT. Plates are again washed with wash buffer and blotted on paper towels.

Add 100  $\mu$ l/well of Enhancement Solution. Shake for 5 minutes. Read the plate on the Wallac DELFIA Fluorometer. Readings from triplicate samples in each assay were tabulated and averaged.

A positive result in this assay suggests AoSMC cell proliferation and that the polypeptide of the present invention may be involved in dermal fibroblast proliferation and/or smooth muscle cell proliferation. A positive result also suggests many potential uses of polypeptides, polynucleotides, agonists and/or antagonists of the polynucleotide/polypeptide of the present invention which gives a positive result. For example, inflammation and immune responses, wound healing, and angiogenesis, as detailed throughout this specification. Particularly, polypeptides of the present invention and polynucleotides of the present invention may be used in wound healing and dermal regeneration, as well as the promotion of vasculogenesis, both of the blood vessels and lymphatics. The growth of vessels can be used in the treatment of, for example, cardiovascular diseases. Additionally, antagonists of polypeptides and polynucleotides of the invention may be useful in treating diseases, disorders, and/or conditions which involve angiogenesis by acting as an anti-vascular agent (e.g., anti-angiogenesis). These diseases, disorders, and/or conditions are known in the art and/or are described herein, such as, for example, malignancies, solid tumors,

benign tumors, for example hemangiomas, acoustic neuromas, neurofibromas, trachomas, and pyogenic granulomas; artheroscleric plaques; ocular angiogenic diseases, for example, diabetic retinopathy, retinopathy of prematurity, macular degeneration, corneal graft rejection, neovascular glaucoma, retrolental fibroplasia, rubeosis, retinoblastoma, uvietis and Pterygia (abnormal blood vessel growth) of the eye; rheumatoid arthritis; psoriasis; delayed wound healing; endometriosis; vasculogenesis; granulations; hypertrophic scars (keloids); nonunion fractures; scleroderma; trachoma; vascular adhesions; myocardial angiogenesis; coronary collaterals; cerebral collaterals; arteriovenous malformations; ischemic limb angiogenesis; Osler-Webber Syndrome; plaque neovascularization; telangiectasia; hemophiliac joints; angiofibroma; fibromuscular dysplasia; wound granulation; Crohn's disease; and atherosclerosis. Moreover, antagonists of polypeptides and polynucleotides of the invention may be useful in treating anti-hyperproliferative diseases and/or anti-inflammatory known in the art and/or described herein.

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One skilled in the art could easily modify the exemplified studies to test the activity of polynucleotides (e.g., gene therapy), antibodies, agonists, and/or antagonists and fragments and variants thereof.

### Example 39: Cellular Adhesion Molecule (CAM) Expression on Endothelial Cells

The recruitment of lymphocytes to areas of inflammation and angiogenesis involves specific receptor-ligand interactions between cell surface adhesion molecules (CAMs) on lymphocytes and the vascular endothelium. The adhesion process, in both normal and pathological settings, follows a multi-step cascade that involves intercellular adhesion molecule-1 (ICAM-1), vascular cell adhesion molecule-1 (VCAM-1), and endothelial leukocyte adhesion molecule-1 (E-selectin) expression on endothelial cells (EC). The expression of these molecules and others on the vascular endothelium determines the efficiency with which leukocytes may adhere to the local vasculature and extravasate into the local tissue during the development of an inflammatory response. The local concentration of cytokines and growth factor participate in the modulation of the expression of these CAMs.

Briefly, endothelial cells (e.g., Human Umbilical Vein Endothelial cells (HUVECs)) are grown in a standard 96 well plate to confluence, growth medium is removed from the cells and replaced with 100  $\mu$ l of 199 Medium (10% fetal bovine serum (FBS)). Samples for testing and positive or negative controls are added to the plate in triplicate (in 10  $\mu$ l volumes). Plates are then incubated at 37°C for either 5 h (selectin and integrin expression) or 24 h (integrin expression only). Plates are aspirated to remove medium and 100  $\mu$ l of 0.1% paraformaldehyde-PBS(with Ca++ and Mg++) is added to each well. Plates are held at 4°C for 30 min. Fixative is removed from the wells and wells are washed 1X with PBS(+Ca,Mg) + 0.5% BSA and drained. 10  $\mu$ l of

diluted primary antibody is added to the test and control wells. Anti-ICAM-1-Biotin, Anti-VCAM-1-Biotin and Anti-E-selectin-Biotin are used at a concentration of 10 μg/ml (1:10 dilution of 0.1 mg/ml stock antibody). Cells are incubated at 37°C for 30 min. in a humidified environment. Wells are washed three times with PBS(+Ca,Mg) + 0.5% BSA. 20 µl of diluted ExtrAvidin-Alkaline Phosphatase (1:5,000 dilution, referred to herein as the working dilution) are added to each well and incubated at 37°C for 30 min. Wells are washed three times with PBS(+Ca,Mg)+0.5% BSA. Dissolve 1 tablet of p-Nitrophenol Phosphate pNPP per 5 ml of glycine buffer (pH 10.4). 100 µl of pNPP substrate in glycine buffer is added to each test well. Standard wells in triplicate are prepared from the working dilution of the ExtrAvidin-Alkaline Phosphotase in glycine buffer: 1:5,000 ( $10^{0}$ ) >  $10^{-0.5}$  >  $10^{-1}$  >  $10^{-1.5}$ . 5  $\mu$ l of each dilution is added to triplicate wells and the resulting AP content in each well is 5.50 ng, 1.74 ng, 0.55 ng, 0.18 ng. 100 μl of pNNP reagent is then added to each of the standard wells. The plate is incubated at 37°C for 4h. A volume of 50  $\mu$ l of 3M NaOH is added to all wells. The plate is read on a plate reader at 405 nm using the background subtraction option on blank wells filled with glycine buffer only. Additionally, the template is set up to indicate the concentration of AP-conjugate in each standard well [5.50 ng; 1.74 ng; 0.55 ng; 0.18 ng]. Results are indicated as amount of bound AP-conjugate in each sample.

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# Example 40: Alamar Blue Endothelial Cells Proliferation Assay

This assay may be used to quantitatively determine protein mediated inhibition of bFGF-induced proliferation of Bovine Lymphatic Endothelial Cells (LECs), Bovine Aortic Endothelial Cells (BAECs) or Human Microvascular Uterine Myometrial Cells (UTMECs). This assay incorporates a fluorometric growth indicator based on detection of metabolic activity. A standard Alamar Blue Proliferation Assay is prepared in EGM-2MV with 10 ng /ml of bFGF added as a source of endothelial cell stimulation. This assay may be used with a variety of endothelial cells with slight changes in growth medium and cell concentration. Dilutions of the protein batches to be tested are diluted as appropriate. Serum-free medium (GIBCO SFM) without bFGF is used as a non-stimulated control and Angiostatin or TSP-1 are included as a known inhibitory controls.

Briefly, LEC, BAECs or UTMECs are seeded in growth media at a density of 5000 to 2000 cells/well in a 96 well plate and placed at 37 degreesC overnight. After the overnight incubation of the cells, the growth media is removed and replaced with GIBCO EC-SFM. The cells are treated with the appropriate dilutions of the protein of interest or control protein sample(s) (prepared in SFM) in triplicate wells with additional bFGF to a concentration of 10 ng/ml. Once the cells have been treated with the samples, the plate(s) is/are placed back in the 37° C incubator for three days. After three days 10 ml of stock alamar blue (Biosource Cat# DAL1100) is added to

each well and the plate(s) is/are placed back in the 37°C incubator for four hours. The plate(s) are then read at 530nm excitation and 590nm emission using the CytoFluor fluorescence reader. Direct output is recorded in relative fluorescence units.

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Alamar blue is an oxidation-reduction indicator that both fluoresces and changes color in response to chemical reduction of growth medium resulting from cell growth. As cells grow in culture, innate metabolic activity results in a chemical reduction of the immediate surrounding environment. Reduction related to growth causes the indicator to change from oxidized (non-fluorescent blue) form to reduced (fluorescent red) form (i.e., stimulated proliferation will produce a stronger signal and inhibited proliferation will produce a weaker signal and the total signal is proportional to the total number of cells as well as their metabolic activity). The background level of activity is observed with the starvation medium alone. This is compared to the output observed from the positive control samples (bFGF in growth medium) and protein dilutions.

#### Example 41: Detection of Inhibition of a Mixed Lymphocyte Reaction

This assay can be used to detect and evaluate inhibition of a Mixed Lymphocyte Reaction (MLR) by gene products (e.g., isolated polypeptides). Inhibition of a MLR may be due to a direct effect on cell proliferation and viability, modulation of costimulatory molecules on interacting cells, modulation of adhesiveness between lymphocytes and accessory cells, or modulation of cytokine production by accessory cells. Multiple cells may be targeted by these polypeptides since the peripheral blood mononuclear fraction used in this assay includes T, B and natural killer lymphocytes, as well as monocytes and dendritic cells.

Polypeptides of interest found to inhibit the MLR may find application in diseases associated with lymphocyte and monocyte activation or proliferation. These include, but are not limited to, diseases such as asthma, arthritis, diabetes, inflammatory skin conditions, psoriasis, eczema, systemic lupus erythematosus, multiple sclerosis, glomerulonephritis, inflammatory bowel disease, crohn's disease, ulcerative colitis, arteriosclerosis, cirrhosis, graft vs. host disease, host vs. graft disease, hepatitis, leukemia and lymphoma.

Briefly, PBMCs from human donors are purified by density gradient centrifugation using Lymphocyte Separation Medium (LSM[®], density 1.0770 g/ml, Organon Teknika Corporation, West Chester, PA). PBMCs from two donors are adjusted to 2 x 10⁶ cells/ml in RPMI-1640 (Life Technologies, Grand Island, NY) supplemented with 10% FCS and 2 mM glutamine. PBMCs from a third donor is adjusted to 2 x 10⁵ cells/ml. Fifty microliters of PBMCs from each donor is added to wells of a 96-well round bottom microtiter plate. Dilutions of test materials (50 μl) is added in triplicate to microtiter wells. Test samples (of the protein of interest) are added for final dilution of 1:4; rhuIL-2 (R&D Systems, Minneapolis, MN, catalog number 202-IL) is added to a

final concentration of 1  $\mu$ g/ml; anti-CD4 mAb (R&D Systems, clone 34930.11, catalog number MAB379) is added to a final concentration of 10  $\mu$ g/ml. Cells are cultured for 7-8 days at 37°C in 5% CO₂, and 1  $\mu$ C of [³H] thymidine is added to wells for the last 16 hrs of culture. Cells are harvested and thymidine incorporation determined using a Packard TopCount. Data is expressed as the mean and standard deviation of triplicate determinations.

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Samples of the protein of interest are screened in separate experiments and compared to the negative control treatment, anti-CD4 mAb, which inhibits proliferation of lymphocytes and the positive control treatment, IL-2 (either as recombinant material or supernatant), which enhances proliferation of lymphocytes.

One skilled in the art could easily modify the exemplified studies to test the activity of polynucleotides (e.g., gene therapy), antibodies, agonists, and/or antagonists and fragments and variants thereof.

#### Example 42: Assays for Protease Activity

The following assay may be used to assess protease activity of the polypeptides of the invention.

Gelatin and casein zymography are performed essentially as described (Heusen et al., Anal. Biochem., 102:196-202 (1980); Wilson et al., Journal of Urology, 149:653-658 (1993)). Samples are run on 10% polyacryamide/0.1% SDS gels containing 1% gelain orcasein, soaked in 2.5% triton at room temperature for 1 hour, and in 0.1M glycine, pH 8.3 at 37°C 5 to 16 hours. After staining in amido black areas of proteolysis apear as clear areas agains the blue-black background. Trypsin (Sigma T8642) is used as a positive control.

Protease activity is also determined by monitoring the cleavage of n-a-benzoyl-L-arginine ethyl ester (BAEE) (Sigma B-4500. Reactions are set up in (25mMNaPO₄,1mM EDTA, and 1mM BAEE), pH 7.5. Samples are added and the change in adsorbance at 260nm is monitored on the Beckman DU-6 spectrophotometer in the time-drive mode. Trypsin is used as a positive control.

Additional assays based upon the release of acid-soluble peptides from casein or hemoglobin measured as adsorbance at 280 nm or colorimetrically using the Folin method are performed as described in Bergmeyer, et al., *Methods of Enzymatic Analysis*, 5 (1984). Other assays involve the solubilization of chromogenic substrates (Ward, *Applied Science*, 251-317 (1983)).

#### Example 43: Identifying Serine Protease Substrate Specificity

Methods known in the art or described herein may be used to determine the substrate specificity of the polypeptides of the present invention having serine protease activity. A preferred

method of determining substrate specificity is by the use of positional scanning synthetic combinatorial libraries as described in GB 2 324 529 (incorporated herein in its entirety).

## Example 44: Ligand Binding Assays

The following assay may be used to assess ligand binding activity of the polypeptides of the invention.

Ligand binding assays provide a direct method for ascertaining receptor pharmacology and are adaptable to a high throughput format. The purified ligand for a polypeptide is radiolabeled to high specific activity (50-2000 Ci/mmol) for binding studies. A determination is then made that the process of radiolabeling does not diminish the activity of the ligand towards its polypeptide. Assay conditions for buffers, ions, pH and other modulators such as nucleotides are optimized to establish a workable signal to noise ratio for both membrane and whole cell polypeptide sources. For these assays, specific polypeptide binding is defined as total associated radioactivity minus the radioactivity measured in the presence of an excess of unlabeled competing ligand. Where possible, more than one competing ligand is used to define residual nonspecific binding.

#### Example 45: Functional Assay in Xenopus Oocytes

Capped RNA transcripts from linearized plasmid templates encoding the polypeptides of the invention are synthesized in vitro with RNA polymerases in accordance with standard procedures. In vitro transcripts are suspended in water at a final concentration of 0.2 mg/mi. Ovarian lobes are removed from adult female toads, Stage V defolliculated oocytes are obtained, and RNA transcripts (10 ng/oocytc) are injected in a 50 nl bolus using a microinjection apparatus. Two electrode voltage clamps are used to measure the currents from individual Xenopus oocytes in response polypeptides and polypeptide agonist exposure. Recordings are made in Ca2+ free Barth's medium at room temperature. The Xenopus system can be used to screen known ligands and tissue/cell extracts for activating ligands.

#### Example 46: Microphysiometric Assays

Activation of a wide variety of secondary messenger systems results in extrusion of small amounts of acid from a cell. The acid formed is largely as a result of the increased metabolic activity required to fuel the intracellular signaling process. The pH changes in the media surrounding the cell are very small but are detectable by the CYTOSENSOR microphysiometer (Molecular Devices Ltd., Menlo Park, Calif.). The CYTOSENSOR is thus capable of detecting the activation of polypeptide which is coupled to an energy utilizing intracellular signaling pathway.

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#### Example 47: Extract/Cell Supernatant Screening

A large number of mammalian receptors exist for which there remains, as yet, no cognate activating ligand (agonist). Thus, active ligands for these receptors may not be included within the ligands banks as identified to date. Accordingly, the polypeptides of the invention can also be functionally screened (using calcium, cAMP, microphysiometer, oocyte electrophysiology, etc., functional screens) against tissue extracts to identify its natural ligands. Extracts that produce positive functional responses can be sequentially subfractionated until an activating ligand is isolated and identified.

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#### Example 48: Calcium and cAMP Functional Assays

Seven transmembrane receptors which are expressed in HEK 293 cells have been shown to be coupled functionally to activation of PLC and calcium mobilization and/or cAMP stimulation or inhibition. Basal calcium levels in the HEK 293 cells in receptor-transfected or vector control cells were observed to be in the normal, 100 nM to 200 nM, range. HEK 293 cells expressing recombinant receptors are loaded with fura 2 and in a single day >150 selected ligands or tissue/cell extracts are evaluated for agonist induced calcium mobilization. Similarly, HEK 293 cells expressing recombinant receptors are evaluated for the stimulation or inhibition of cAMP production using standard cAMP quantitation assays. Agonists presenting a calcium transient or cAMP fluctuation are tested in vector control cells to determine if the response is unique to the transfected cells expressing receptor.

#### Example 49: ATP-binding assay

The following assay may be used to assess ATP-binding activity of polypeptides of the invention.

ATP-binding activity of the polypeptides of the invention may be detected using the ATP-binding assay described in U.S. Patent 5,858,719, which is herein incorporated by reference in its entirety. Briefly, ATP-binding to polypeptides of the invention is measured via photoaffinity labeling with 8-azido-ATP in a competition assay. Reaction mixtures containing 1 mg/ml of the ABC transport protein of the present invention are incubated with varying concentrations of ATP, or the non-hydrolyzable ATP analog adenyl-5'-imidodiphosphate for 10 minutes at 4°C. A mixture of 8-azido-ATP (Sigma Chem. Corp., St. Louis, MO.) plus 8-azido-ATP (³²P-ATP) (5 mCi/μmol, ICN, Irvine CA.) is added to a final concentration of 100 μM and 0.5 ml aliquots are placed in the wells of a porcelain spot plate on ice. The plate is irradiated using a short wave 254 nm UV lamp at a distance of 2.5 cm from the plate for two one-minute intervals with a one-minute cooling interval in between. The reaction is stopped by addition of dithiothreitol to a final concentration of

2mM. The incubations are subjected to SDS-PAGE electrophoresis, dried, and autoradiographed. Protein bands corresponding to the particular polypeptides of the invention are excised, and the radioactivity quantified. A decrease in radioactivity with increasing ATP or adenly-5'-imidodiphosphate provides a measure of ATP affinity to the polypeptides.

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# Example 50: Small Molecule Screening

This invention is particularly useful for screening therapeutic compounds by using the polypeptides of the invention, or binding fragments thereof, in any of a variety of drug screening techniques. The polypeptide or fragment employed in such a test may be affixed to a solid support, expressed on a cell surface, free in solution, or located intracellularly. One method of drug screening utilizes eukaryotic or prokaryotic host cells which are stably transformed with recombinant nucleic acids expressing the polypeptide or fragment. Drugs are screened against such transformed cells in competitive binding assays. One may measure, for example, the formulation of complexes between the agent being tested and polypeptide of the invention.

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Thus, the present invention provides methods of screening for drugs or any other agents which affect activities mediated by the polypeptides of the invention. These methods comprise contacting such an agent with a polypeptide of the invention or fragment thereof and assaying for the presence of a complex between the agent and the polypeptide or fragment thereof, by methods well known in the art. In such a competitive binding assay, the agents to screen are typically labeled. Following incubation, free agent is separated from that present in bound form, and the amount of free or uncomplexed label is a measure of the ability of a particular agent to bind to the polypeptides of the invention.

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Another technique for drug screening provides high throughput screening for compounds having suitable binding affinity to the polypeptides of the invention, and is described in great detail in European Patent Application 84/03564, published on September 13, 1984, which is herein incorporated by reference in its entirety. Briefly stated, large numbers of different small molecule test compounds are synthesized on a solid substrate, such as plastic pins or some other surface. The test compounds are reacted with polypeptides of the invention and washed. Bound polypeptides are then detected by methods well known in the art. Purified polypeptides are coated directly onto plates for use in the aforementioned drug screening techniques. In addition, non-neutralizing antibodies may be used to capture the peptide and immobilize it on the solid support.

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This invention also contemplates the use of competitive drug screening assays in which neutralizing antibodies capable of binding polypeptides of the invention specifically compete with a test compound for binding to the polypeptides or fragments thereof. In this manner, the

antibodies are used to detect the presence of any peptide which shares one or more antigenic epitopes with a polypeptide of the invention.

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## Example 51: Phosphorylation Assay

In order to assay for phosphorylation activity of the polypeptides of the invention, a phosphorylation assay as described in U.S. Patent 5,958,405 (which is herein incorporated by reference) is utilized. Briefly, phosphorylation activity may be measured by phosphorylation of a protein substrate using gamma-labeled ³²P-ATP and quantitation of the incorporated radioactivity using a gamma radioisotope counter. The polypeptides of the invention are incubated with the protein substrate, ³²P-ATP, and a kinase buffer. The ³²P incorporated into the substrate is then separated from free ³²P-ATP by electrophoresis, and the incorporated ³²P is counted and compared to a negative control. Radioactivity counts above the negative control are indicative of phosphorylation activity of the polypeptides of the invention.

# Example 52: Detection of Phosphorylation Activity (Activation) of the Polypeptides of the Invention in the Presence of Polypeptide Ligands

Methods known in the art or described herein may be used to determine the phosphorylation activity of the polypeptides of the invention. A preferred method of determining phosphorylation activity is by the use of the tyrosine phosphorylation assay as described in US 5,817,471 (incorporated herein by reference).

# Example 53: Identification Of Signal Transduction Proteins That Interact With Polypeptides Of The Present Invention

The purified polypeptides of the invention are research tools for the identification, characterization and purification of additional signal transduction pathway proteins or receptor proteins. Briefly, labeled polypeptides of the invention are useful as reagents for the purification of molecules with which it interacts. In one embodiment of affinity purification, polypeptides of the invention are covalently coupled to a chromatography column. Cell-free extract derived from putative target cells, such as carcinoma tissues, is passed over the column, and molecules with appropriate affinity bind to the polypeptides of the invention. The protein complex is recovered from the column, dissociated, and the recovered molecule subjected to N-terminal protein sequencing. This amino acid sequence is then used to identify the captured molecule or to design degenerate oligonucleotide probes for cloning the relevant gene from an appropriate cDNA library.

#### Example 54: IL-6 Bioassay

To test the proliferative effects of the polypeptides of the invention, the IL-6 Bioassay as described by Marz et al. is utilized (Proc. Natl. Acad. Sci., U.S.A., 95:3251-56 (1998), which is herein incorporated by reference). Briefly, IL-6 dependent B9 murine cells are washed three times in IL-6 free medium and plated at a concentration of 5,000 cells per well in 50  $\mu$ l, and 50  $\mu$ l of the IL-6-like polypeptide is added. After 68 hrs. at 37°C, the number of viable cells is measured by adding the tetrazolium salt thiazolyl blue (MTT) and incubating for a further 4 hrs. at 37°C. B9 cells are lysed by SDS and optical density is measured at 570 nm. Controls containing IL-6 (positive) and no cytokine (negative) are utilized. Enhanced proliferation in the test sample(s) relative to the negative control is indicative of proliferative effects mediated by polypeptides of the invention.

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### Example 55: Assay for Phosphatase Activity

The following assay may be used to assess serine/threonine phosphatase (PTPase) activity of the polypeptides of the invention.

In order to assay for serine/threonine phosphatase (PTPase) activity, assays can be utilized which are widely known to those skilled in the art. For example, the serine/threonine phosphatase (PSPase) activity is measured using a PSPase assay kit from New England Biolabs, Inc. Myelin basic protein (MyBP), a substrate for PSPase, is phosphorylated on serine and threonine residues with cAMP-dependent Protein Kinase in the presence of [32P]ATP. Protein serine/threonine phosphatase activity is then determined by measuring the release of inorganic phosphate from 32P-labeled MyBP.

#### Example 56: Interaction of Serine/Threonine Phosphatases with other Proteins

The polypeptides of the invention with serine/threonine phosphatase activity as determined in Example 58 are research tools for the identification, characterization and purification of additional interacting proteins or receptor proteins, or other signal transduction pathway proteins. Briefly, labeled polypeptide(s) of the invention is useful as a reagent for the purification of molecules with which it interacts. In one embodiment of affinity purification, polypeptide of the invention is covalently coupled to a chromatography column. Cell-free extract derived from putative target cells, such as neural or liver cells, is passed over the column, and molecules with appropriate affinity bind to the polypeptides of the invention. The polypeptides of the invention complex is recovered from the column, dissociated, and the recovered molecule subjected to N-terminal protein sequencing. This amino acid sequence is then used to identify the captured

molecule or to design degenerate oligonucleotide probes for cloning the relevant gene from an appropriate cDNA library.

## Example 57: Assaying for Heparanase Activity

In order to assay for heparanase activity of the polypeptides of the invention, the heparanase assay described by Vlodavsky et al is utilized (Vlodavsky, I., et al., Nat. Med., 5:793-802 (1999)). Briefly, cell lysates, conditioned media or intact cells (1 x  $10^6$  cells per 35-mm dish) are incubated for 18 hrs at 37°C, pH 6.2-6.6, with  35 S-labeled ECM or soluble ECM derived peak I proteoglycans. The incubation medium is centrifuged and the supernatant is analyzed by gel filtration on a Sepharose CL-6B column (0.9 x 30 cm). Fractions are eluted with PBS and their radioactivity is measured. Degradation fragments of heparan sulfate side chains are eluted from Sepharose 6B at 0.5 <  $K_{av}$  < 0.8 (peak II). Each experiment is done at least three times. Degradation fragments corresponding to "peak II," as described by Vlodavsky et al., is indicative of the activity of the polypeptides of the invention in cleaving heparan sulfate.

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#### Example 58: Immobilization of biomolecules

This example provides a method for the stabilization of polypeptides of the invention in non-host cell lipid bilayer constucts (see, e.g., Bieri et al., Nature Biotech 17:1105-1108 (1999), hereby incorporated by reference in its entirety herein) which can be adapted for the study of polypeptides of the invention in the various functional assays described above. Briefly, carbohydrate-specific chemistry for biotinylation is used to confine a biotin tag to the extracellular domain of the polypeptides of the invention, thus allowing uniform orientation upon immobilization. A 50uM solution of polypeptides of the invention in washed membranes is incubated with 20 mM NaIO4 and 1.5 mg/ml (4mM) BACH or 2 mg/ml (7.5mM) biotinhydrazide for 1 hr at room temperature (reaction volume, 150ul). Then the sample is dialyzed (Pierce Slidealizer Cassett, 10 kDa cutoff; Pierce Chemical Co., Rockford IL) at 4C first for 5 h, exchanging the buffer after each hour, and finally for 12 h against 500 ml buffer R (0.15 M NaCl, 1 mM MgCl2, 10 mM sodium phosphate, pH7). Just before addition into a cuvette, the sample is diluted 1:5 in buffer ROG50 (Buffer R supplemented with 50 mM octylglucoside).

# Example 59: TAQMAN

Quantitative PCR (QPCR). Total RNA from cells in culture are extracted by Trizol separation as recommended by the supplier (LifeTechnologies). (Total RNA is treated with DNase I (Life Technologies) to remove any contaminating genomic DNA before reverse transcription.) Total RNA (50 ng) is used in a one-step, 50ul, RT-QPCR, consisting of Taqman Buffer A (Perkin-

Elmer; 50 mM KCl/10 mM Tris, pH 8.3), 5.5 mM MgCl₂, 240 μM each dNTP, 0.4 units RNase inhibitor(Promega), 8%glycerol, 0.012% Tween-20, 0.05% gelatin, 0.3uM primers, 0.1uM probe, 0.025units Amplitaq Gold (Perkin-Elmer) and 2.5 units Superscript II reverse transcriptase (Life Technologies). As a control for genomic contamination, parallel reactions are setup without reverse transcriptase. The relative abundance of (unknown) and 18S RNAs are assessed by using the Applied Biosystems Prism 7700 Sequence Detection System (Livak, K. J., Flood, S. J., Marmaro, J., Giusti, W. & Deetz, K. (1995) PCR Methods Appl. 4, 357-362). Reactions are carried out at 48°C for 30 min, 95°C for 10 min, followed by 40 cycles of 95°C for 15s, 60°C for 1 min. Reactions are performed in triplicate.

Primers (f & r) and FRET probes sets are designed using Primer Express Software (Perkin-Elmer). Probes are labeled at the 5'-end with the reporter dye 6-FAM and on the 3'-end with the quencher dye TAMRA (Biosource International, Camarillo, CA or Perkin-Elmer).

## Example 60: Assays for Metalloproteinase Activity

Metalloproteinases (EC 3.4.24.-) are peptide hydrolases which use metal ions, such as Zn²⁺, as the catalytic mechanism. Metalloproteinase activity of polypeptides of the present invention can be assayed according to the following methods.

#### Proteolysis of alpha-2-macroglobulin

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To confirm protease activity, purified polypeptides of the invention are mixed with the substrate alpha-2-macroglobulin (0.2 unit/ml; Boehringer Mannheim, Germany) in 1x assay buffer (50 mM HEPES, pH 7.5, 0.2 M NaCl, 10 mM CaCl₂, 25 µM ZnCl₂ and 0.05% Brij-35) and incubated at 37°C for 1-5 days. Trypsin is used as positive control. Negative controls contain only alpha-2-macroglobulin in assay buffer. The samples are collected and boiled in SDS-PAGE sample buffer containing 5% 2-mercaptoethanol for 5-min, then loaded onto 8% SDS-polyacrylamide gel. After electrophoresis the proteins are visualized by silver staining. Proteolysis is evident by the appearance of lower molecular weight bands as compared to the negative control.

# Inhibition of alpha-2-macroglobulin proteolysis by inhibitors of metalloproteinases

Known metalloproteinase inhibitors (metal chelators (EDTA, EGTA, AND HgCl₂), peptide metalloproteinase inhibitors (TIMP-1 and TIMP-2), and commercial small molecule MMP inhibitors) are used to characterize the proteolytic activity of polypeptides of the invention. The three synthetic MMP inhibitors used are: MMP inhibitor I, [IC₅₀ = 1.0  $\mu$ M against MMP-1 and MMP-8; IC₅₀ = 30  $\mu$ M against MMP-9; IC₅₀ = 150  $\mu$ M against MMP-3]; MMP-3 (stromelysin-1) inhibitor I [IC₅₀ = 5  $\mu$ M against MMP-3], and MMP-3 inhibitor II [K_i = 130 nM against MMP-3];

inhibitors available through Calbiochem, catalog # 444250, 444218, and 444225, respectively). Briefly, different concentrations of the small molecule MMP inhibitors are mixed with purified polypeptides of the invention (50µg/ml) in 22.9 µl of 1x HEPES buffer (50 mM HEPES, pH 7.5, 0.2 M NaCl, 10 mM CaCl₂, 25 µM ZnCl₂ and 0.05%Brij-35) and incubated at room temperature (24 °C) for 2-hr, then 7.1 µl of substrate alpha-2-macroglobulin (0.2 unit/ml) is added and incubated at 37°C for 20-hr. The reactions are stopped by adding 4x sample buffer and boiled immediately for 5 minutes. After SDS-PAGE, the protein bands are visualized by silver stain.

#### Synthetic Fluorogenic Peptide Substrates Cleavage Assay

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The substrate specificity for polypeptides of the invention with demonstrated metalloproteinase activity can be determined using synthetic fluorogenic peptide substrates (purchased from BACHEM Bioscience Inc). Test substrates include, M-1985, M-2225, M-2105, M-2110, and M-2255. The first four are MMP substrates and the last one is a substrate of tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) converting enzyme (TACE). All the substrates are prepared in 1:1 dimethyl sulfoxide (DMSO) and water. The stock solutions are 50-500  $\mu$ M. Fluorescent assays are performed by using a Perkin Elmer LS 50B luminescence spectrometer equipped with a constant temperature water bath. The excitation  $\lambda$  is 328 nm and the emission  $\lambda$  is 393 nm. Briefly, the assay is carried out by incubating 176  $\mu$ l 1x HEPES buffer (0.2 M NaCl, 10 mM CaCl₂, 0.05% Brij-35 and 50 mM HEPES, pH 7.5) with 4  $\mu$ l of substrate solution (50  $\mu$ M) at 25 °C for 15 minutes, and then adding 20  $\mu$ l of a purified polypeptide of the invention into the assay cuvett. The final concentration of substrate is 1  $\mu$ M. Initial hydrolysis rates are monitored for 30-min.

# Example 61: Characterization of the cDNA contained in a deposited plasmid

The size of the cDNA insert contained in a deposited plasmid may be routinely determined using techniques known in the art, such as PCR amplification using synthetic primers hybridizable to the 3' and 5' ends of the cDNA sequence. For example, two primers of 17-30 nucleotides derived from each end of the cDNA (i.e., hybridizable to the absolute 5' nucleotide or the 3' nucleotide end of the sequence of SEQ ID NO:X, respectively) are synthesized and used to amplify the cDNA using the deposited cDNA plasmid as a template. The polymerase chain reaction is carried out under routine conditions, for instance, in 25 ul of reaction mixture with 0.5 ug of the above cDNA template. A convenient reaction mixture is 1.5-5 mM MgCl₂, 0.01% (w/v) gelatin, 20 uM each of dATP, dCTP, dGTP, dTTP, 25 pmol of each primer and 0.25 Unit of Taq polymerase. Thirty five cycles of PCR (denaturation at 94 degree C for 1 min; annealing at 55 degree C for 1 min; elongation at 72 degree C for 1 min) are performed with a Perkin-Elmer Cetus

automated thermal cycler. The amplified product is analyzed by agarose gel electrophoresis. The PCR product is verified to be the selected sequence by subcloning and sequencing the DNA product. It will be clear that the invention may be practiced otherwise than as particularly described in the foregoing description and examples. Numerous modifications and variations of the present invention are possible in light of the above teachings and, therefore, are within the scope of the appended claims.

#### Incorporation by Reference

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The entire disclosure of each document cited (including patents, patent applications, journal articles, abstracts, laboratory manuals, books, or other disclosures) in the Background of the Invention, Detailed Description, and Examples is hereby incorporated herein by reference. In addition, the sequence listing submitted herewith is incorporated herein by reference in its entirety. The specification and sequence listing of each of the following U.S. and PCT applications are herein incorporated by reference in their entirety: U.S. Appln. No. 60/040,162 filed on 07-Mar-1997, U.S. Appln. No. 60/043,576 filed on 11-Apr-1997, U.S. Appln. No. 60/047,601 filed on 23-May-1997, U.S. Appln. No. 60/056,845 filed on 22-Aug-1997, U.S. Appln. No. 60/043,580 filed on 11-Apr-1997, U.S. Appln. No. 60/047,599 filed on 23-May-1997, U.S. Appln. No. 60/056,664 filed on 22-Aug-1997, U.S. Appln. No. 60/043,314 filed on 11-Apr-1997, U.S. Appln. No. 60/047,632 filed on 23-May-1997, U.S. Appln. No. 60/056,892 filed on 22-Aug-1997, U.S. Appln. No. 60/043.568 filed on 11-Apr-1997, U.S. Appln. No. 60/047,595 filed on 23-May-1997, U.S. Appln. No. 60/056,632 filed on 22-Aug-1997, U.S. Appln. No. 60/043,578 filed on 11-Apr-1997, U.S. Appln. No. 60/040,333 filed on 07-Mar-1997, U.S. Appln. No. 60/043,670 filed on 11-Apr-1997, U.S. Appln. No. 60/047,596 filed on 23-May-1997, U.S. Appln. No. 60/056,864 filed on 22-Aug-1997, U.S. Appln. No. 60/043,674 filed on 11-Apr-1997, U.S. Appln. No. 60/047,612 filed on 23-May-1997, U.S. Appln. No. 60/056,631 filed on 22-Aug-1997, U.S. Appln. No. 60/043,569 filed on 11-Apr-1997, U.S. Appln. No. 60/047,588 filed on 23-May-1997, U.S. Appln. No. 60/056,876 filed on 22-Aug-1997, U.S. Appln. No. 60/043,671 filed on 11-Apr-1997, U.S. Appln. No. 60/043,311 filed on 11-Apr-1997, U.S. Appln. No. 60/038,621 filed on 07-Mar-1997, U.S. Appln. No. 60/043,672 filed on 11-Apr-1997, U.S. Appln. No. 60/047,613 filed on 23-May-1997, U.S. Appln. No. 60/056,636 filed on 22-Aug-1997, U.S. Appln. No. 60/043,669 filed on 11-Apr-1997, U.S. Appln. No. 60/047,582 filed on 23-May-1997, U.S. Appln. No. 60/056,910 filed on 22-Aug-1997, U.S. Appln. No. 60/043,315 filed on 11-Apr-1997, U.S. Appln. No. 60/047,598 filed on 23-May-1997, U.S. Appln. No. 60/056,874 filed on 22-Aug-1997, U.S. Appln. No. 60/043,312 filed on 11-Apr-1997, U.S. Appln. No. 60/047,585 filed on 23-May-1997, U.S. Appln. No.

60/056,881 filed on 22-Aug-1997, U.S. Appln. No. 60/043,313 filed on 11-Apr-1997, U.S. Appln. No. 60/047,586 filed on 23-May-1997, U.S. Appln. No. 60/056,909 filed on 22-Aug-1997, U.S. Appln. No. 60/040,161 filed on 07-Mar-1997, U.S. Appln. No. 60/047,587 filed on 23-May-1997, U.S. Appln. No. 60/056,879 filed on 22-Aug-1997, U.S. Appln. No. 60/047,500 filed on 23-May-1997, U.S. Appln. No. 60/056,880 filed on 22-Aug-1997, U.S. Appln. No. 60/047,584 filed on 23-May-1997, U.S. Appln. No. 60/056,894 filed on 22-Aug-1997, U.S. Appln. No. 60/047,492 filed on 23-May-1997, U.S. Appln. No. 60/056,911 filed on 22-Aug-1997, U.S. Appln. No. 60/040,626 filed on 07-Mar-1997, U.S. Appln. No. 60/047,503 filed on 23-May-1997, U.S. Appln. No. 60/056,903 filed on 22-Aug-1997, U.S. Appln. No. 60/047,501 filed on 23-May-1997, U.S. Appln. No. 60/056,637 filed on 22-Aug-1997, U.S. Appln. No. 60/047,590 filed on 23-May-1997, U.S. 10 Appln. No. 60/056,875 filed on 22-Aug-1997, U.S. Appln. No. 60/047,581 filed on 23-May-1997, U.S. Appln. No. 60/056,882 filed on 22-Aug-1997, U.S. Appln. No. 60/047,592 filed on 23-May-1997, U.S. Appln. No. 60/056,888 filed on 22-Aug-1997, U.S. Appln. No. 60/040,334 filed on 07-Mar-1997, U.S. Appln. No. 60/047,618 filed on 23-May-1997, U.S. Appln. No. 60/056,872 filed on 22-Aug-1997, U.S. Appln. No. 60/047,617 filed on 23-May-1997, U.S. Appln. No. 60/056,662 15 filed on 22-Aug-1997, U.S. Appln. No. 60/047,589 filed on 23-May-1997, U.S. Appln. No. 60/056,862 filed on 22-Aug-1997, U.S. Appln. No. 60/047,594 filed on 23-May-1997, U.S. Appln. No. 60/056,884 filed on 22-Aug-1997, U.S. Appln. No. 60/047,583 filed on 23-May-1997, U.S. Appln. No. 60/056,878 filed on 22-Aug-1997, U.S. Appln. No. 60/040,336 filed on 07-Mar-1997, U.S. Appln. No. 60/047,502 filed on 23-May-1997, U.S. Appln. No. 60/056,893 filed on 22-Aug-20 1997, U.S. Appln. No. 60/047,633 filed on 23-May-1997, U.S. Appln. No. 60/056,630 filed on 22-Aug-1997, U.S. Appln. No. 60/047,593 filed on 23-May-1997, U.S. Appln. No. 60/056,887 filed on 22-Aug-1997, U.S. Appln. No. 60/040,163 filed on 07-Mar-1997, U.S. Appln. No. 60/047,597 filed on 23-May-1997, U.S. Appln. No. 60/056,889 filed on 22-Aug-1997, U.S. Appln. No. 60/047,615 filed on 23-May-1997, U.S. Appln. No. 60/056,877 filed on 22-Aug-1997, U.S. Appln. 25 No. 60/047,600 filed on 23-May-1997, U.S. Appln. No. 60/056,886 filed on 22-Aug-1997, U.S. Appln. No. 60/047,614 filed on 23-May-1997, U.S. Appln. No. 60/056,908 filed on 22-Aug-1997, U.S. Appln. No. 60/040,710 filed on 14-Mar-1997, U.S. Appln. No. 60/050,934 filed on 30-May-1997, U.S. Appln. No. 60/048,100 filed on 30-May-1997, U.S. Appln. No. 60/040,762 filed on 14-Mar-1997, U.S. Appln. No. 60/048,357 filed on 30-May-1997, U.S. Appln. No. 60/048,189 filed 30 on 30-May-1997, U.S. Appln. No. 60/041,277 filed on 21-Mar-1997, U.S. Appln. No. 60/048,188 filed on 30-May-1997, U.S. Appln. No. 60/048,094 filed on 30-May-1997, U.S. Appln. No. 60/048,350 filed on 30-May-1997, U.S. Appln. No. 60/048,135 filed on 30-May-1997, U.S. Appln. No. 60/042,344 filed on 21-Mar-1997, U.S. Appln. No. 60/048,187 filed on 30-May-1997, U.S. Appln. No. 60/048,099 filed on 30-May-1997, U.S. Appln. No. 60/050,937 filed on 30-May-35

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1997, U.S. Appln. No. 60/048,352 filed on 30-May-1997, U.S. Appln. No. 60/041,276 filed on 21-Mar-1997, U.S. Appln. No. 60/048,069 filed on 30-May-1997, U.S. Appln. No. 60/048,131 filed on 30-May-1997, U.S. Appln. No. 60/048,186 filed on 30-May-1997, U.S. Appln. No. 60/048,095 filed on 30-May-1997, U.S. Appln. No. 60/041,281 filed on 21-Mar-1997, U.S. Appln. No. 60/048,355 filed on 30-May-1997, U.S. Appln. No. 60/048,096 filed on 30-May-1997, U.S. Appln. No. 60/048,351 filed on 30-May-1997, U.S. Appln. No. 60/048,154 filed on 30-May-1997, U.S. Appln. No. 60/048,160 filed on 30-May-1997, U.S. Appln. No. 60/042,825 filed on 08-Apr-1997, U.S. Appln. No. 60/048,070 filed on 30-May-1997, U.S. Appln. No. 60/042,727 filed on 08-Apr-1997, U.S. Appln. No. 60/048,068 filed on 30-May-1997, U.S. Appln. No. 60/042,726 filed on 08-Apr-1997, U.S. Appln. No. 60/048,184 filed on 30-May-1997, U.S. Appln. No. 60/042,728 filed on 08-Apr-1997, U.S. Appln. No. 60/042,754 filed on 08-Apr-1997, U.S. Appln. No. 60/048,190 filed on 30-May-1997, U.S. Appln. No. 60/044,039 filed on 30-May-1997, U.S. Appln. No. 60/048,093 filed on 30-May-1997, U.S. Appln. No. 60/048,885 filed on 06-Jun-1997, U.S. Appln. No. 60/057,645 filed on 05-Sep-1997, U.S. Appln. No. 60/049,375 filed on 06-Jun-1997, U.S. Appln. No. 60/057,642 filed on 05-Sep-1997, U.S. Appln. No. 60/048,881 filed on 06-Jun-1997, U.S. Appln. No. 60/057,668 filed on 05-Sep-1997, U.S. Appln. No. 60/048,880 filed on 06-Jun-1997, U.S. Appln. No. 60/057,635 filed on 05-Sep-1997, U.S. Appln. No. 60/048,896 filed on 06-Jun-1997, U.S. Appln. No. 60/057,627 filed on 05-Sep-1997, U.S. Appln. No. 60/049,020 filed on 06-Jun-1997, U.S. Appln. No. 60/057,667 filed on 05-Sep-1997, U.S. Appln. No. 60/048.876 filed on 06-Jun-1997, U.S. Appln. No. 60/057,666 filed on 05-Sep-1997, U.S. Appln. No. 60/048.895 filed on 06-Jun-1997, U.S. Appln. No. 60/057,764 filed on 05-Sep-1997, U.S. Appln. No. 60/048,884 filed on 06-Jun-1997, U.S. Appln. No. 60/057,643 filed on 05-Sep-1997, U.S. Appln. No. 60/048,894 filed on 06-Jun-1997, U.S. Appln. No. 60/057,769 filed on 05-Sep-1997, U.S. Appln. No. 60/048,971 filed on 06-Jun-1997, U.S. Appln. No. 60/057,763 filed on 05-Sep-1997, U.S. Appln. No. 60/048,964 filed on 06-Jun-1997, U.S. Appln. No. 60/057,650 filed on 05-Sep-1997, U.S. Appln. No. 60/048,882 filed on 06-Jun-1997, U.S. Appln. No. 60/057,584 filed on 05-Sep-1997, U.S. Appln. No. 60/048,899 filed on 06-Jun-1997, U.S. Appln. No. 60/057,647 filed on 05-Sep-1997, U.S. Appln. No. 60/048,893 filed on 06-Jun-1997, U.S. Appln. No. 60/057,661 filed on 05-Sep-1997, U.S. Appln. No. 60/048,900 filed on 06-Jun-1997, U.S. Appln. No. 60/057,662 filed on 05-Sep-1997, U.S. Appln. No. 60/048,901 filed on 06-Jun-1997, U.S. Appln. No. 60/057,646 filed on 05-Sep-1997, U.S. Appln. No. 60/048,892 filed on 06-Jun-1997, U.S. Appln. No. 60/057,654 filed on 05-Sep-1997, U.S. Appln. No. 60/048,915 filed on 06-Jun-1997, U.S. Appln. No. 60/057,651 filed on 05-Sep-1997, U.S. Appln. No. 60/049,019 filed on 06-Jun-1997, U.S. Appln. No. 60/057,644 filed on 05-Sep-1997, U.S. Appln. No. 60/048,970 filed on 06-Jun-1997, U.S. Appln. No. 60/057,765 filed on 05-Sep-1997, U.S. Appln. No. 60/048,972 filed

on 06-Jun-1997, U.S. Appln. No. 60/057,762 filed on 05-Sep-1997, U.S. Appln. No. 60/048,916 filed on 06-Jun-1997, U.S. Appln. No. 60/057,775 filed on 05-Sep-1997, U.S. Appln. No. 60/049,373 filed on 06-Jun-1997, U.S. Appln. No. 60/057,648 filed on 05-Sep-1997, U.S. Appln. No. 60/048,875 filed on 06-Jun-1997, U.S. Appln. No. 60/057,774 filed on 05-Sep-1997, U.S. Appln. No. 60/049,374 filed on 06-Jun-1997, U.S. Appln. No. 60/057,649 filed on 05-Sep-1997, 5 U.S. Appln. No. 60/048,917 filed on 06-Jun-1997, U.S. Appln. No. 60/057,770 filed on 05-Sep-1997, U.S. Appln. No. 60/048,949 filed on 06-Jun-1997, U.S. Appln. No. 60/057,771 filed on 05-Sep-1997, U.S. Appln. No. 60/048,974 filed on 06-Jun-1997, U.S. Appln. No. 60/057,761 filed on 05-Sep-1997, U.S. Appln. No. 60/048,883 filed on 06-Jun-1997, U.S. Appln. No. 60/057,760 filed on 05-Sep-1997, U.S. Appln. No. 60/048,897 filed on 06-Jun-1997, U.S. Appln. No. 60/057,776 10 filed on 05-Sep-1997, U.S. Appln. No. 60/048,898 filed on 06-Jun-1997, U.S. Appln. No. 60/057,778 filed on 05-Sep-1997, U.S. Appln. No. 60/048,962 filed on 06-Jun-1997, U.S. Appln. No. 60/057,629 filed on 05-Sep-1997, U.S. Appln. No. 60/048,963 filed on 06-Jun-1997, U.S. Appln. No. 60/057,628 filed on 05-Sep-1997, U.S. Appln. No. 60/048,877 filed on 06-Jun-1997, U.S. Appln. No. 60/057,777 filed on 05-Sep-1997, U.S. Appln. No. 60/048,878 filed on 06-Jun-15 1997, U.S. Appln. No. 60/057,634 filed on 05-Sep-1997, U.S. Appln. No. 60/049,608 filed on 13-Jun-1997, U.S. Appln. No. 60/058,669 filed on 12-Sep-1997, U.S. Appln. No. 60/049,566 filed on 13-Jun-1997, U.S. Appln. No. 60/058,668 filed on 12-Sep-1997, U.S. Appln. No. 60/052,989 filed on 13-Jun-1997, U.S. Appln. No. 60/058,750 filed on 12-Sep-1997, U.S. Appln. No. 60/049,607 filed on 13-Jun-1997, U.S. Appln. No. 60/058,665 filed on 12-Sep-1997, U.S. Appln. No. 20 60/049,611 filed on 13-Jun-1997, U.S. Appln. No. 60/058,971 filed on 12-Sep-1997, U.S. Appln. No. 60/050,901 filed on 13-Jun-1997, U.S. Appln. No. 60/058,972 filed on 12-Sep-1997, U.S. Appln. No. 60/049,609 filed on 13-Jun-1997, U.S. Appln. No. 60/058,975 filed on 12-Sep-1997, U.S. Appln. No. 60/048,356 filed on 30-May-1997, U.S. Appln. No. 60/056,296 filed on 29-Aug-1997, U.S. Appln. No. 60/048,101 filed on 30-May-1997, U.S. Appln. No. 60/056,293 filed on 29-25 Aug-1997, U.S. Appln. No. 60/050,935 filed on 30-May-1997, U.S. Appln. No. 60/056,250 filed on 29-Aug-1997, U.S. Appln. No. 60/049,610 filed on 13-Jun-1997, U.S. Appln. No. 60/061,060 filed on 02-Oct-1997, U.S. Appln. No. 60/049,606 filed on 13-Jun-1997, U.S. Appln. No. 60/060,841 filed on 02-Oct-1997, U.S. Appln. No. 60/049,550 filed on 13-Jun-1997, U.S. Appln. No. 60/060,834 filed on 02-Oct-1997, U.S. Appln. No. 60/049,549 filed on 13-Jun-1997, U.S. 30 Appln. No. 60/060,865 filed on 02-Oct-1997, U.S. Appln. No. 60/049,548 filed on 13-Jun-1997, U.S. Appln. No. 60/060,844 filed on 02-Oct-1997, U.S. Appln. No. 60/049,547 filed on 13-Jun-1997, U.S. Appln. No. 60/061,059 filed on 02-Oct-1997, U.S. Appln. No. 60/051,381 filed on 01-Jul-1997, U.S. Appln. No. 60/058,598 filed on 12-Sep-1997, U.S. Appln. No. 60/051,480 filed on 01-Jul-1997, U.S. Appln. No. 60/058,663 filed on 12-Sep-1997, U.S. Appln. No. 60/051,926 filed 35

on 08-Jul-1997, U.S. Appln. No. 60/058,785 filed on 12-Sep-1997, U.S. Appln. No. 60/052,793 filed on 08-Jul-1997, U.S. Appln. No. 60/058,664 filed on 12-Sep-1997, U.S. Appln. No. 60/051,925 filed on 08-Jul-1997, U.S. Appln. No. 60/058,660 filed on 12-Sep-1997, U.S. Appln. No. 60/051,929 filed on 08-Jul-1997, U.S. Appln. No. 60/058,661 filed on 12-Sep-1997, U.S. Appln. No. 60/052,803 filed on 08-Jul-1997, U.S. Appln. No. 60/055,722 filed on 18-Aug-1997, U.S. Appln. No. 60/052,732 filed on 08-Jul-1997, U.S. Appln. No. 60/055,723 filed on 18-Aug-1997, U.S. Appln. No. 60/051,932 filed on 08-Jul-1997, U.S. Appln. No. 60/055,948 filed on 18-Aug-1997, U.S. Appln. No. 60/051,931 filed on 08-Jul-1997, U.S. Appln. No. 60/055,949 filed on 18-Aug-1997, U.S. Appln. No. 60/051,916 filed on 08-Jul-1997, U.S. Appln. No. 60/055,953 filed on 18-Aug-1997, U.S. Appln. No. 60/051;930 filed on 08-Jul-1997, U.S. Appln. No. 60/055,950 filed on 18-Aug-1997, U.S. Appln. No. 60/051,918 filed on 08-Jul-1997, U.S. Appln. No. 60/055,947 filed on 18-Aug-1997, U.S. Appln. No. 60/051,920 filed on 08-Jul-1997, U.S. Appln. No. 60/055,964 filed on 18-Aug-1997, U.S. Appln. No. 60/052,733 filed on 08-Jul-1997, U.S. Appln. No. 60/056,360 filed on 18-Aug-1997, U.S. Appln. No. 60/052,795 filed on 08-Jul-1997, U.S. Appln. No. 60/055,684 filed on 18-Aug-1997, U.S. Appln. No. 60/051,919 filed on 08-Jul-1997, U.S. Appln. No. 60/055,984 filed on 18-Aug-1997, U.S. Appln. No. 60/051,928 filed on 08-Jul-1997, U.S. Appln. No. 60/055,954 filed on 18-Aug-1997, U.S. Appln. No. 60/052,870 filed on 16-Jul-1997, U.S. Appln. No. 60/055,952 filed on 18-Aug-1997, U.S. Appln. No. 60/052,871 filed on 16-Jul-1997, U.S. Appln. No. 60/055,725 filed on 18-Aug-1997, U.S. Appln. No. 60/052,872 filed on 16-Jul-1997, U.S. Appln. No. 60/056,359 filed on 18-Aug-1997, U.S. Appln. No. 60/052,661 filed on 16-Jul-1997, U.S. Appln. No. 60/055,985 filed on 18-Aug-1997, U.S. Appln. No. 60/052,874 filed on 16-Jul-1997, U.S. Appln. No. 60/055,724 filed on 18-Aug-1997, U.S. Appln. No. 60/052,873 filed on 16-Jul-1997, U.S. Appln. No. 60/055,726 filed on 18-Aug-1997, U.S. Appln. No. 60/052,875 filed on 16-Jul-1997, U.S. Appln. No. 60/056,361 filed on 18-Aug-1997, U.S. Appln. No. 60/053,440 filed on 22-Jul-1997, U.S. Appln. No. 60/055,989 filed on 18-Aug-1997, U.S. Appln. No. 60/053,441 filed on 22-Jul-1997, U.S. Appln. No. 60/055,946 filed on 18-Aug-1997, U.S. Appln. No. 60/053,442 filed on 22-Jul-1997, U.S. Appln. No. 60/055,683 filed on 18-Aug-1997, U.S. Appln. No. 60/054,212 filed on 30-Jul-1997, U.S. Appln. No. 60/055,968 filed on 18-Aug-1997, U.S. Appln. No. 60/054,209 filed on 30-Jul-1997, U.S. Appln. No. 60/055,972 filed on 18-Aug-1997, U.S. Appln. No. 60/054,234 filed on 30-Jul-1997, U.S. Appln. No. 60/055,969 filed on 18-Aug-1997, U.S. Appln. No. 60/055,386 filed on 05-Aug-1997, U.S. Appln. No. 60/055,986 filed on 18-Aug-1997, U.S. Appln. No. 60/054,807 filed on 05-Aug-1997, U.S. Appln. No. 60/055,970 filed on 18-Aug-1997, U.S. Appln. No. 60/054,215 filed on 30-Jul-1997, U.S. Appln. No. 60/056,543 filed on 19-Aug-1997, U.S. Appln. No. 60/054,218 filed on 30-Jul-1997, U.S. Appln. No. 60/056,561 filed on 19-Aug-1997, U.S. Appln. No. 60/054,214 filed on

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30-Jul-1997, U.S. Appln. No. 60/056,534 filed on 19-Aug-1997, U.S. Appln. No. 60/054,236 filed on 30-Jul-1997, U.S. Appln. No. 60/056,729 filed on 19-Aug-1997, U.S. Appln. No. 60/054,213 filed on 30-Jul-1997, U.S. Appln. No. 60/056,727 filed on 19-Aug-1997, U.S. Appln. No. 60/054,211 filed on 30-Jul-1997, U.S. Appln. No. 60/056,554 filed on 19-Aug-1997, U.S. Appln. No. 60/054,217 filed on 30-Jul-1997, U.S. Appln. No. 60/056,730 filed on 19-Aug-1997, U.S. Appln. No. 60/055,312 filed on 05-Aug-1997, U.S. Appln. No. 60/056,563 filed on 19-Aug-1997, U.S. Appln. No. 60/055,309 filed on 05-Aug-1997, U.S. Appln. No. 60/056,557 filed on 19-Aug-1997, U.S. Appln. No. 60/055,310 filed on 05-Aug-1997, U.S. Appln. No. 60/056,371 filed on 19-Aug-1997, U.S. Appln. No. 60/054,798 filed on 05-Aug-1997, U.S. Appln. No. 60/056,732 filed on 19-Aug-1997, U.S. Appln. No. 60/056,369 filed on 19-Aug-1997, U.S. Appln. No. 60/056,535 filed on 19-Aug-1997, U.S. Appln. No. 60/056,556 filed on 19-Aug-1997, U.S. Appln. No. 60/056,555 filed on 19-Aug-1997, U.S. Appln. No. 60/054,806 filed on 05-Aug-1997, U.S. Appln. No. 60/056,366 filed on 19-Aug-1997, U.S. Appln. No. 60/054,809 filed on 05-Aug-1997, U.S. Appln. No. 60/056,364 filed on 19-Aug-1997, U.S. Appln. No. 60/054,804 filed on 05-Aug-1997, U.S. Appln. No. 60/056,370 filed on 19-Aug-1997, U.S. Appln. No. 60/054,803 filed on 05-Aug-1997, U.S. Appln. No. 60/056,731 filed on 19-Aug-1997, U.S. Appln. No. 60/055,311 filed on 05-Aug-1997, U.S. Appln. No. 60/056,365 filed on 19-Aug-1997, U.S. Appln. No. 60/054,808 filed on 05-Aug-1997, U.S. Appln. No. 60/056,367 filed on 19-Aug-1997, U.S. Appln. No. 60/056,726 filed on 19-Aug-1997, U.S. Appln. No. 60/056,368 filed on 19-Aug-1997, U.S. Appln. No. 60/056,728 filed on 19-Aug-1997, U.S. Appln. No. 60/056,628 filed on 19-Aug-1997, U.S. Appln. No. 60/056,629 filed on 19-Aug-1997, U.S. Appln. No. 60/056,270 filed on 29-Aug-1997, U.S. Appln. No. 60/056,271 filed on 29-Aug-1997, U.S. Appln. No. 60/056,247 filed on 29-Aug-1997, U.S. Appln. No. 60/056,073 filed on 29-Aug-1997, U.S. Appln. No. 60/057,669 filed on 05-Sep-1997, U.S. Appln. No. 60/057,663 filed on 05-Sep-1997, U.S. Appln. No. 60/057,626 filed on 05-Sep-1997, U.S. Appln. No. 60/058,666 filed on 12-Sep-1997, U.S. Appln. No. 60/058,973 filed on 12-Sep-1997, U.S. Appln. No. 60/058,974 filed on 12-Sep-1997, U.S. Appln. No. 60/058,667 filed on 12-Sep-1997, U.S. Appln. No. 60/060,837 filed on 02-Oct-1997, U.S. Appln. No. 60/060,862 filed on 02-Oct-1997, U.S. Appln. No. 60/060,839 filed on 02-Oct-1997, U.S. Appln. No. 60/060,866 filed on 02-Oct-1997, U.S. Appln. No. 60/060,843 filed on 02-Oct-1997, U.S. Appln. No. 60/060,836 filed on 02-Oct-1997, U.S. Appln. No. 60/060,838 filed on 02-Oct-1997, U.S. Appln. No. 60/060,874 filed on 02-Oct-1997, U.S. Appln. No. 60/060,833 filed on 02-Oct-1997, U.S. Appln. No. 60/060,884 filed on 02-Oct-1997, U.S. Appln. No. 60/060,880 filed on 02-Oct-1997, U.S. Appln. No. 60/061,463 filed on 09-Oct-1997, U.S. Appln. No. 60/061,529 filed on 09-Oct-1997, U.S. Appln. No. 60/071,498 filed on 09-Oct-1997, U.S. Appln. No. 60/061,527 filed on 09-Oct-1997, U.S. Appln. No. 60/061,536 filed on 09-Oct-1997, U.S. Appln. No. 60/061,532 filed

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on 09-Oct-1997, U.S. Appln. No. 60/063,099 filed on 24-Oct-1997, U.S. Appln. No. 60/063,088 filed on 24-Oct-1997, U.S. Appln. No. 60/063,100 filed on 24-Oct-1997, U.S. Appln. No. 60/063,387 filed on 24-Oct-1997, U.S. Appln. No. 60/063,148 filed on 24-Oct-1997, U.S. Appln. No. 60/063,386 filed on 24-Oct-1997, U.S. Appln. No. 60/062,784 filed on 24-Oct-1997, U.S. Appln. No. 60/063,091 filed on 24-Oct-1997, U.S. Appln. No. 60/063,090 filed on 24-Oct-1997, U.S. Appln. No. 60/063,089 filed on 24-Oct-1997, U.S. Appln. No. 60/063,092 filed on 24-Oct-1997, U.S. Appln. No. 60/063,111 filed on 24-Oct-1997, U.S. Appln. No. 60/063,101 filed on 24-Oct-1997, U.S. Appln. No. 60/063,109 filed on 24-Oct-1997, U.S. Appln. No. 60/063,110 filed on 24-Oct-1997, U.S. Appln. No. 60/063,098 filed on 24-Oct-1997, U.S. Appln. No. 60/063,097 filed on 24-Oct-1997, U.S. Appln. No. 60/064,911 filed on 07-Nov-1997, U.S. Appln. No. 60/064,912 10 filed on 07-Nov-1997, U.S. Appln. No. 60/064,983 filed on 07-Nov-1997, U.S. Appln. No. 60/064,900 filed on 07-Nov-1997, U.S. Appln. No. 60/064,988 filed on 07-Nov-1997, U.S. Appln. No. 60/064,987 filed on 07-Nov-1997, U.S. Appln. No. 60/064,908 filed on 07-Nov-1997, U.S. Appln. No. 60/064,984 filed on 07-Nov-1997, U.S. Appln. No. 60/064,985 filed on 07-Nov-1997, U.S. Appln. No. 60/066,094 filed on 17-Nov-1997, U.S. Appln. No. 60/066,100 filed on 17-Nov-15 1997, U.S. Appln. No. 60/066,089 filed on 17-Nov-1997, U.S. Appln. No. 60/066,095 filed on 17-Nov-1997, U.S. Appln. No. 60/066,090 filed on 17-Nov-1997, U.S. Appln. No. 60/068,006 filed on 18-Dec-1997, U.S. Appln. No. 60/068,057 filed on 18-Dec-1997, U.S. Appln. No. 60/068,007 filed on 18-Dec-1997, U.S. Appln. No. 60/068,008 filed on 18-Dec-1997, U.S. Appln. No. 20 60/068,054 filed on 18-Dec-1997, U.S. Appln. No. 60/068,064 filed on 18-Dec-1997, U.S. Appln. No. 60/068,053 filed on 18-Dec-1997, U.S. Appln. No. 60/070,923 filed on 18-Dec-1997, U.S. Appln. No. 60/068,365 filed on 19-Dec-1997, U.S. Appln. No. 60/068,169 filed on 19-Dec-1997, U.S. Appln. No. 60/068,367 filed on 19-Dec-1997, U.S. Appln. No. 60/068,369 filed on 19-Dec-1997, U.S. Appln. No. 60/068,368 filed on 19-Dec-1997, U.S. Appln. No. 60/070,657 filed on 07-Jan-1998, U.S. Appln. No. 60/070,692 filed on 07-Jan-1998, U.S. Appln. No. 60/070,704 filed on 25 07-Jan-1998, U.S. Appln. No. 60/070,658 filed on 07-Jan-1998, U.S. Appln. No. 60/073,160 filed on 30-Jan-1998, U.S. Appln. No. 60/073,159 filed on 30-Jan-1998, U.S. Appln. No. 60/073,165 filed on 30-Jan-1998, U.S. Appln. No. 60/073,164 filed on 30-Jan-1998, U.S. Appln. No. 60/073,167 filed on 30-Jan-1998, U.S. Appln. No. 60/073,162 filed on 30-Jan-1998, U.S. Appln. No. 60/073,161 filed on 30-Jan-1998, U.S. Appln. No. 60/073,170 filed on 30-Jan-1998, U.S. 30 Appln. No. 60/074,141 filed on 09-Feb-1998, U.S. Appln. No. 60/074,341 filed on 09-Feb-1998, U.S. Appln. No. 60/074,037 filed on 09-Feb-1998, U.S. Appln. No. 60/074,157 filed on 09-Feb-1998, U.S. Appln. No. 60/074,118 filed on 09-Feb-1998, U.S. Appln. No. 60/076,051 filed on 26-Feb-1998, U.S. Appln. No. 60/076,053 filed on 26-Feb-1998, U.S. Appln. No. 60/076,054 filed on 26-Feb-1998, U.S. Appln. No. 60/076,052 filed on 26-Feb-1998, U.S. Appln. No. 60/076,057 filed 35

on 26-Feb-1998, U.S. Appln. No. 60/077,714 filed on 12-Mar-1998, U.S. Appln. No. 60/077,687 filed on 12-Mar-1998, U.S. Appln. No. 60/077,686 filed on 12-Mar-1998, U.S. Appln. No. 60/077,696 filed on 12-Mar-1998, U.S. Appln. No. 60/078,566 filed on 19-Mar-1998, U.S. Appln. No. 60/078,574 filed on 19-Mar-1998, U.S. Appln. No. 60/078,576 filed on 19-Mar-1998, U.S. Appln. No. 60/078,579 filed on 19-Mar-1998, U.S. Appln. No. 60/078,563 filed on 19-Mar-1998, U.S. Appln. No. 60/078,573 filed on 19-Mar-1998, U.S. Appln. No. 60/078,578 filed on 19-Mar-1998, U.S. Appln. No. 60/078,581 filed on 19-Mar-1998, U.S. Appln. No. 60/078,577 filed on 19-Mar-1998, U.S. Appln. No. 60/080,314 filed on 01-Apr-1998, U.S. Appln. No. 60/080,312 filed on 01-Apr-1998, U.S. Appln. No. 60/080,313 filed on 01-Apr-1998, U.S. Appln. No. 60/085,180 filed on 12-May-1998, U.S. Appln. No. 60/085,105 filed on 12-May-1998, U.S. Appln. No. 60/085,094 filed on 12-May-1998, U.S. Appln. No. 60/085,093 filed on 12-May-1998, U.S. Appln. No. 60/085,924 filed on 18-May-1998, U.S. Appln. No. 60/085,906 filed on 18-May-1998, U.S. Appln. No. 60/085,927 filed on 18-May-1998, U.S. Appln. No. 60/085,920 filed on 18-May-1998, U.S. Appln. No. 60/085,928 filed on 18-May-1998, U.S. Appln. No. 60/085,925 filed on 18-May-1998, U.S. Appln. No. 60/085,921 filed on 18-May-1998, U.S. Appln. No. 60/085,923 filed on 18-May-1998, U.S. Appln. No. 60/085,922 filed on 18-May-1998, U.S. Appln. No. 60/090,112 filed on 22-Jun-1998, U.S. Appln. No. 60/089,508 filed on 16-Jun-1998, U.S. Appln. No. 60/089,507 filed on 16-Jun-1998, U.S. Appln. No. 60/089,510 filed on 16-Jun-1998, U.S. Appln. No. 60/089,509 filed on 16-Jun-1998, U.S. Appln. No. 60/090,113 filed on 22-Jun-1998, U.S. Appln. No. 60/092,956 filed on 15-Jul-1998, U.S. Appln. No. 60/092,921 filed on 15-Jul-1998, U.S. Appln. No. 60/092,922 filed on 15-Jul-1998, U.S. Appln. No. 60/094,657 filed on 30-Jul-1998, U.S. Appln. No. 60/095,486 filed on 05-Aug-1998, U.S. Appln. No. 60/096,319 filed on 12-Aug-1998, U.S. Appln. No. 60/095,455 filed on 06-Aug-1998, U.S. Appln. No. 60/095,454 filed on 06-Aug-1998, U.S. Appln. No. 60/097,917 filed on 25-Aug-1998, U.S. Appln. No. 60/098,634 filed on 31-Aug-1998, U.S. Appln. No. 60/101,546 filed on 23-Sep-1998, U.S. Appln. No. 60/102,895 filed on 02-Oct-1998, U.S. Appln. No. 60/108,207 filed on 12-Nov-1998, U.S. Appln. No. 60/113,006 filed on 18-Dec-1998, U.S. Appln. No. 60/112,809 filed on 17-Dec-1998, U.S. Appln. No. 60/116,330 filed on 19-Jan-1999, U.S. Appln. No. 60/119,468 filed on 10-Feb-1999, U.S. Appln. No. 60/125,055 filed on 18-Mar-1999, U.S. Appln. No. 60/128,693 filed on 09-Apr-1999, U.S. Appln. No. 60/130,991 filed on 26-Apr-1999, U.S. Appln. No. 60/137,725 filed on 07-Jun-1999, U.S. Appln. No. 60/145,220 filed on 23-Jul-1999, U.S. Appln. No. 60/149,182 filed on 17-Aug-1999, U.S. Appln. No. 60/152,317 filed on 03-Sep-1999, U.S. Appln. No. 60/152,315 filed on 03-Sep-1999, U.S. Appln. No. 60/155,709 filed on 24-Sep-1999, U.S. Appln. No. 60/163,085 filed on 02-Nov-1999, U.S. Appln. No. 60/172,411 filed on 17-Dec-1999, U.S. Appln. No. 60/162,239 filed on 29-Oct-1999, U.S. Appln. No. 60/215,139 filed on 30-Jun-2000, U.S. Appln. No.

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60/162,211 filed on 29-Oct-1999, U.S. Appln. No. 60/215,138 filed on 30-Jun-2000, U.S. Appln. No. 60/162,240 filed on 29-Oct-1999, U.S. Appln. No. 60/215,131 filed on 30-Jun-2000, U.S. Appln. No. 60/162,237 filed on 29-Oct-1999, U.S. Appln. No. 60/219,666 filed on 21-Jul-2000, U.S. Appln. No. 60/162,238 filed on 29-Oct-1999, U.S. Appln. No. 60/215,134 filed on 30-Jun-2000, U.S. Appln. No. 60/163,580 filed on 05-Nov-1999, U.S. Appln. No. 60/215,130 filed on 30-Jun-2000, U.S. Appln. No. 60/163,577 filed on 05-Nov-1999, U.S. Appln. No. 60/215,137 filed on 30-Jun-2000, U.S. Appln. No. 60/163,581 filed on 05-Nov-1999, U.S. Appln. No. 60/215,133 filed on 30-Jun-2000, U.S. Appln. No. 60/163,576 filed on 05-Nov-1999, U.S. Appln. No. 60/221,366 filed on 27-Jul-2000, U.S. Appln. No. 60/164,344 filed on 09-Nov-1999, U.S. Appln. No. 60/195,296 filed on 07-Apr-2000, U.S. Appln. No. 60/221,367 filed on 27-Jul-2000, U.S. Appln. No. 60/164,835 filed on 12-Nov-1999, U.S. Appln. No. 60/221,142 filed on 27-Jul-2000, U.S. Appln. No. 60/164,744 filed on 12-Nov-1999, U.S. Appln. No. 60/215,140 filed on 30-Jun-2000, U.S. Appln. No. 60/164,735 filed on 12-Nov-1999, U.S. Appln. No. 60/221,193 filed on 27-Jul-2000, U.S. Appln. No. 60/164,825 filed on 12-Nov-1999, U.S. Appln. No. 60/222,904 filed on 03-Aug-2000, U.S. Appln. No. 60/164,834 filed on 12-Nov-1999, U.S. Appln. No. 60/224,007 filed 15 on 04-Aug-2000, U.S. Appln. No. 60/164,750 filed on 12-Nov-1999, U.S. Appln. No. 60/215,128 filed on 30-Jun-2000, U.S. Appln. No. 60/166,415 filed on 19-Nov-1999, U.S. Appln. No. 60/215,136 filed on 30-Jun-2000, U.S. Appln. No. 60/166,414 filed on 19-Nov-1999, U.S. Appln. No. 60/219,665 filed on 21-Jul-2000, U.S. Appln. No. 60/164,731 filed on 12-Nov-1999, U.S. Appln. No. 60/215,132 filed on 30-Jun-2000, U.S. Appln. No. 60/226,280 filed on 18-Aug-2000, 20 U.S. Appln. No. 60/256,968 filed on 21-Dec-2000, U.S. Appln. No. 60/226,380 filed on 18-Aug-2000, U.S. Appln. No. 60/259,803 filed on 05-Jan-2001, U.S. Appln. No. 60/228,084 filed on 28-Aug-2000, U.S. Appln. No. 09/915,582 filed on 27-Jul-2001, U.S. Appln. No. 60/231,968 filed on 12-Sep-2000, U.S. Appln. No. 60/236,326 filed on 29-Sep-2000, U.S. Appln. No. 60/234,211 filed on 20-Sep-2000, U.S. Appln. No. 60/226,282 filed on 18-Aug-2000, U.S. Appln. No. 60/232,104 25 filed on 12-Sep-2000, U.S. Appln. No. 60/234,210 filed on 20-Sep-2000, U.S. Appln. No. 60/226,278 filed on 18-Aug-2000, U.S. Appln. No. 60/259,805 filed on 05-Jan-2001, U.S. Appln. No. 60/226,279 filed on 18-Aug-2000, U.S. Appln. No. 60/259,678 filed on 05-Jan-2001, U.S. Appln. No. 60/226,281 filed on 18-Aug-2000, U.S. Appln. No. 60/231,969 filed on 12-Sep-2000, U.S. Appln. No. 60/228,086 filed on 28-Aug-2000, U.S. Appln. No. 60/259,516 filed on 04-Jan-30 2001, U.S. Appln. No. 60/228,083 filed on 28-Aug-2000, U.S. Appln. No. 60/259,804 filed on 05-Jan-2001, U.S. Appln. No. 60/270,658 filed on 23-Feb-2001, U.S. Appln. No. 60/304,444 filed on 12-Jul-2001, U.S. Appln. No. 60/270,625 filed on 23-Feb-2001, U.S. Appln. No. 60/304,417 filed on 12-Jul-2001, U.S. Appln. No. 60/295,869 filed on 06-Jun-2001, U.S. Appln. No. 60/304,121 filed on 11-Jul-2001, U.S. Appln. No. 60/311,085 filed on 10-Aug-2001, U.S. Appln. No. 35

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60/325,209 filed on 28-Sep-2001, U.S. Appln. No. 60/330,629 filed on 26-Oct-2001, U.S. Appln. No. 60/331,046 filed on 07-Nov-2001, U.S. Appln. No. 60/358,554 filed on 22-Feb-2002, U.S. Appln. No. 60/358,714 filed on 25-Feb-2002, U.S. Appln. No. 60/277,340 filed on 21-Mar-2001, U.S. Appln. No. 60/306,171 filed on 19-Jul-2001, U.S. Appln. No. 60/278,650 filed on 27-Mar-2001, U.S. Appln. No. 60/331,287 filed on 13-Nov-2001, U.S. Appln. No. 09/950,082 filed on 12-Sep-2001, U.S. Appln. No. 09/950,083 filed on 12-Sep-2001, PCT Appln. No. US00/29363 filed on 25-Oct-2000, PCT Appln. No. US00/29360 filed on 25-Oct-2000, PCT Appln. No. US00/29362 filed on 25-Oct-2000, PCT Appln. No. US00/29365 filed on 25-Oct-2000, PCT Appln. No. US00/29364 filed on 25-Oct-2000, PCT Appln. No. US00/30040 filed on 01-Nov-2000, PCT Appln. No. US00/30037 filed on 01-Nov-2000, PCT Appln. No. US00/30045 filed on 01-Nov-2000, PCT Appln. No. US00/30036 filed on 01-Nov-2000, PCT Appln. No. US00/30039 filed on 01-Nov-2000, PCT Appln. No. US00/30654 filed on 08-Nov-2000, PCT Appln. No. US00/30628 filed on 08-Nov-2000, PCT Appln. No. US00/30653 filed on 08-Nov-2000, PCT Appln. No. US00/30629 filed on 08-Nov-2000, PCT Appln. No. US00/30679 filed on 08-Nov-2000, PCT Appln. No. US00/30674 filed on 08-Nov-2000, PCT Appln. No. US00/31162 filed on 15-Nov-2000, PCT Appln. No. US00/31282 filed on 15-Nov-2000, PCT Appln. No. US00/30657 filed on 08-Nov-2000, PCT Appln. No. US01/01396 filed on 17-Jan-2001, PCT Appln. No. US01/01387 filed on 17-Jan-2001, PCT Appln. No. US01/01567 filed on 17-Jan-2001, PCT Appln. No. US01/01431 filed on 17-Jan-2001, PCT Appln. No. US01/01432 filed on 17-Jan-2001, PCT Appln. No. US01/00544 filed on 09-Jan-2001, PCT Appln. No. US01/01435 filed on 17-Jan-2001, PCT Appln. No. US01/01386 filed on 17-Jan-2001, PCT Appln. No. US01/01565 filed on 17-Jan-2001, PCT Appln. No. US01/01394 filed on 17-Jan-2001, PCT Appln. No. US01/01434 filed on 17-Jan-2001, PCT Appln. No. US01/01397 filed on 17-Jan-2001, PCT Appln. No. US01/01385 filed on 17-Jan-2001, PCT Appln. No. US01/01384 filed on 17-Jan-2001, PCT Appln. No. US01/01383 filed on 17-Jan-2001, PCT Appln. No. (Atty. Dkt. No. PS735; unassigned) filed on 21-Feb-2002, PCT Appln. No. (Atty. Dkt. No. PS736; unassigned) filed on 21-Feb-2002, U.S. Appln. No. 09/148,545 filed on 04-Sep-1998, U.S. Appln. No. 09/621,011 filed on 20-Jul-2000, U.S. Appln. No. 09/981,876 filed on 19-Oct-2001, U.S. Appln. No. 09/149,476 filed on 08-Sep-1998, U.S. Appln. No. 09/809,391 filed on 16-Mar-2001, U.S. Appln. No. 09/882,171 filed on 18-Jun-2001, U.S. Appln. No. 60/190,068 filed on 17-Mar-2000, U.S. Appln. No. 09/152,060 filed on 11-Sep-1998, U.S. Appln. No. 09/852,797 filed on 11-May-2001, U.S. Appln. No. 09/853,161 filed on 11-May-2001, U.S. Appln. No. 09/852,659 filed on 11-May-2001, U.S. Appln. No. 10/058,993 filed on 30-Jan-2002, U.S. Appln. No. 60/265,583 filed on 02-Feb-2001, U.S. Appln. No. 09/154,707 filed on 17-Sep-1998, U.S. Appln. No. 09/966,262 filed on 01-Oct-2001, U.S. Appln. No. 09/983,966 filed on 26-Oct-2001, U.S. Appln. No. 10/059,395 filed on

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31-Jan-2002, U.S. Appln. No. 09/984,245 filed on 29-Oct-2001, U.S. Appln. No. 09/166,780 filed on 06-Oct-1998, U.S. Appln. No. 09/577,145 filed on 24-May-2000, U.S. Appln. No. 09/814,122 filed on 22-Mar-2001, U.S. Appln. No. 09/189,144 filed on 10-Nov-1998, U.S. Appln. No. 09/690,454 filed on 18-Oct-2000, U.S. Appln. No. (Atty. Dkt. No. PZ006G13A; unassigned) filed on 05-Feb-2002, U.S. Appln. No. 10/062,599 filed on 05-Feb-2002, U.S. Appln. No. 09/205,258 filed on 04-Dec-1998, U.S. Appln. No. 09/933,767 filed on 22-Aug-2001, U.S. Appln. No. 60/184,836 filed on 24-Feb-2000, U.S. Appln. No. 60/193,170 filed on 29-Mar-2000, U.S. Appln. No. 10/023,282 filed on 20-Dec-2001, U.S. Appln. No. 10/004,860 filed on 07-Dec-2001, U.S. Appln. No. 09/209,462 filed on 11-Dec-1998, U.S. Appln. No. 09/213,365 filed on 17-Dec-1998, U.S. Appln. No. 09/627,081 filed on 27-Jul-2000, U.S. Appln. No. 09/227,357 filed on 08-Jan-1999, U.S. Appln. No. 09/983,802 filed on 25-Oct-2001, U.S. Appln. No. 09/973,278 filed on 10-Oct-2001, U.S. Appln. No. 60/239,899 filed on 13-Oct-2000, U.S. Appln. No. 09/984,490 filed on 30-Oct-2001, U.S. Appln. No. 09/776,724 filed on 06-Feb-2001, U.S. Appln. No. 09/229,982 filed on 14-Jan-1999, U.S. Appln. No. 09/669,688 filed on 26-Sep-2000, U.S. Appln. No. 60/180,909 filed on 08-Feb-2000, U.S. Appln. No. 09/236,557 filed on 26-Jan-1999, U.S. Appln. No. 09/666,984 filed on 21-Sep-2000, U.S. Appln. No. 09/820,649 filed on 30-Mar-2001, U.S. Appln. No. 60/295,558 filed on 05-Jun-2001, U.S. Appln. No. 09/244,112 filed on 04-Feb-1999, U.S. Appln. No. 09/774,639 filed on 01-Feb-2001, U.S. Appln. No. 09/969,730 filed on 04-Oct-2001, U.S. Appln. No. 60/238,291 filed on 06-Oct-2000, U.S. Appln. No. 09/251,329 filed on 17-Feb-1999, U.S. Appln. No. 09/716,128 filed on 17-Nov-2000, U.S. Appln. No. 09/257,179 filed on 25-Feb-1999, U.S. Appln. No. 09/729,835 filed on 06-Dec-2000, U.S. Appln. No. 09/262,109 filed on 04-Mar-1999, U.S. Appln. No. 09/722,329 filed on 28-Nov-2000, U.S. Appln. No. (Atty. Dkt. No. PZ016P1C1; unassigned) filed on 17-Jan-2002, U.S. Appln. No. 60/262,066 filed on 18-Jan-2001, U.S. Appln. No. 09/281,976 filed on 31-Mar-1999, U.S. Appln. No. 09/288,143 filed on 08-Apr-1999, U.S. Appln. No. 09/984,429 filed on 30-Oct-2001, U.S. Appln. No. 60/244,591 filed on 01-Nov-2000, U.S. Appln. No. 09/296,622 filed on 23-Apr-1999, U.S. Appln. No. 09/305,736 filed on 05-May-1999, U.S. Appln. No. 09/818,683 filed on 28-Mar-2001, U.S. Appln. No. 09/974,879 filed on 12-Oct-2001, U.S. Appln. No. 60/239,893 filed on 13-Oct-2000, U.S. Appln. No. 09/334,595 filed on 17-Jun-1999, U.S. Appln. No. 09/348,457 filed on 07-Jul-1999, U.S. Appln. No. 09/739,907 filed on 20-Dec-2000, U.S. Appln. No. 09/938,671 filed on 27-Aug-2001, U.S. Appln. No. 09/363,044 filed on 29-Jul-1999, U.S. Appln. No. 09/813,153 filed on 21-Mar-2001, U.S. Appln. No. 09/949,925 filed on 12-Sep-2001, U.S. Appln. No. 60/232,150 filed on 12-Sep-2000, U.S. Appln. No. 09/369,247 filed on 05-Aug-1999, U.S. Appln. No. 10/062,548 filed on 05-Feb-2002, U.S. Appln. No. 09/382,572 filed on 25-Aug-1999, U.S. Appln. No. 09/716,129 filed on 17-Nov-2000, U.S. Appln. No. 09/393,022 filed on 09-Sep-1999, U.S. Appln. No. 09/798,889

filed on 06-Mar-2001, U.S. Appln. No. 09/397,945 filed on 17-Sep-1999, U.S. Appln. No. 09/437,658 filed on 10-Nov-1999, U.S. Appln. No. 09/892,877 filed on 28-Jun-2001, U.S. Appln. No. 09/948,783 filed on 10-Sep-2001, U.S. Appln. No. 60/231,846 filed on 11-Sep-2000, U.S. Appln. No. 09/461,325 filed on 14-Dec-1999, U.S. Appln. No. 10/050,873 filed on 18-Jan-2002, U.S. Appln. No. 60/263,230 filed on 23-Jan-2001, U.S. Appln. No. 60/263,681 filed on 24-Jan-2001, U.S. Appln. No. 10/012,542 filed on 12-Dec-2001, U.S. Appln. No. 09/482,273 filed on 13-Jan-2000, U.S. Appln. No. 60/234,925 filed on 25-Sep-2000, U.S. Appln. No. 09/984,276 filed on 29-Oct-2001, U.S. Appln. No. 09/984,271 filed on 29-Oct-2001, U.S. Appln. No. 09/489,847 filed on 24-Jan-2000, U.S. Appln. No. 60/350,898 filed on 25-Jan-2002, U.S. Appln. No. 09/511,554 filed on 23-Feb-2000, U.S. Appln. No. 09/739,254 filed on 19-Dec-2000, U.S. Appln. No. 09/904.615 filed on 16-Jul-2001, U.S. Appln. No. 10/054,988 filed on 25-Jan-2002, U.S. Appln. No. 09/531,119 filed on 20-Mar-2000, U.S. Appln. No. 09/820,893 filed on 30-Mar-2001, U.S. Appln. No. 09/565,391 filed on 05-May-2000, U.S. Appln. No. 09/948,820 filed on 10-Sep-2001, U.S. Appln. No. 09/591,316 filed on 09-Jun-2000, U.S. Appln. No. 09/895,298 filed on 02-Jul-2001, U.S. Appln. No. 09/618,150 filed on 17-Jul-2000, U.S. Appln. No. 09/985,153 filed on 01-Nov-2001, U.S. Appln. No. 09/628,508 filed on 28-Jul-2000, U.S. Appln. No. 09/997,131 filed on 30-Nov-2001, U.S. Appln. No. 09/661,453 filed on 13-Sep-2000, U.S. Appln. No. 10/050,882 filed on 18-Jan-2002, U.S. Appln. No. 09/684,524 filed on 10-Oct-2000, U.S. Appln. No. 10/050,704 filed on 18-Jan-2002, U.S. Appln. No. 09/726,643 filed on 01-Dec-2000, U.S. Appln. No. 10/042,141 filed on 11-Jan-2002, U.S. Appln. No. 09/756,168 filed on 09-Jan-2001, U.S. Appln. No. 09/781,417 filed on 13-Feb-2001, U.S. Appln. No. (Atty. Dkt. No. PZ042P1C1; unassigned) filed on 01-Feb-2002, U.S. Appln. No. 09/789,561 filed on 22-Feb-2001, U.S. Appln. No. 09/800,729 filed on 08-Mar-2001, U.S. Appln. No. 09/832,129 filed on 11-Apr-2001, PCT Appln. No. US98/04482 filed on 06-Mar-1998, PCT Appln. No. US98/04493 filed on 06-Mar-1998, PCT Appln. No.US98/04858 filed on 12-Mar-1998, PCT Appln. No.US98/05311 filed on 19-Mar-1998, PCT Appln. No.US98/06801 filed on 07-Apr-1998, PCT Appln. No.US98/10868 filed on 28-May-1998, PCT Appln. No.US98/11422 filed on 04-Jun-1998, PCT Appln. No.US01/05614 filed on 21-Feb-2001, PCT Appln. No.US98/12125 filed on 11-Jun-1998, PCT Appln. No.US98/13608 filed on 30-Jun-1998, PCT Appln. No.US98/13684 filed on 07-Jul-1998, PCT Appln. No.US98/14613 filed on 15-Jul-1998, PCT Appln. No.US98/15949 filed on 29-Jul-1998, PCT Appln. No. US98/16235 filed on 04-Aug-1998, PCT Appln. No. US98/17044 filed on 18-Aug-1998, PCT Appln. No.US98/17709 filed on 27-Aug-1998, PCT Appln. No.US98/18360 filed on 03-Sep-1998, PCT Appln. No.(Atty. Dkt. No. PZ016PCT2; unassigned) filed on 17-Jan-2002, PCT Appln. No.US98/20775 filed on 01-Oct-1998, PCT Appln. No.US98/21142 filed on 08-Oct-1998, PCT Appln. No.US98/22376 filed on 23-Oct-1998, PCT Appln. No.US98/23435 filed on

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04-Nov-1998, PCT Appln. No.US98/27059 filed on 17-Dec-1998, PCT Appln. No.US99/00108 filed on 06-Jan-1999, PCT Appln. No.US99/01621 filed on 27-Jan-1999, PCT Appln. No.US99/02293 filed on 04-Feb-1999, PCT Appln. No.US99/03939 filed on 24-Feb-1999, PCT Appln. No. US99/05721 filed on 11-Mar-1999, PCT Appln. No. US99/05804 filed on 18-Mar-1999, PCT Appln. No.US99/09847 filed on 06-May-1999, PCT Appln. No.US99/13418 filed on 15-Jun-1999, PCT Appln. No.US99/15849 filed on 14-Jul-1999, PCT Appln. No.US01/00911 filed on 12-Jan-2001, PCT Appln. No.US01/29871 filed on 24-Sep-2001, PCT Appln. No.US99/17130 filed on 29-Jul-1999, PCT Appln. No.US99/19330 filed on 24-Aug-1999, PCT Appln. No.US99/22012 filed on 22-Sep-1999, PCT Appln. No.US99/26409 filed on 09-Nov-1999, PCT Appln. No.US99/29950 filed on 16-Dec-1999, PCT Appln. No.US00/00903 filed on 18-Jan-2000, PCT Appln. No.US00/03062 filed on 08-Feb-2000, PCT Appln. No.US00/06783 filed on 16-Mar-2000, PCT Appln. No.US00/08979 filed on 06-Apr-2000, PCT Appln. No.US00/15187 filed on 02-Jun-2000, PCT Appln. No.US00/19735 filed on 20-Jul-2000, PCT Appln. No.US00/22325 filed on 16-Aug-2000, PCT Appln. No.US00/24008 filed on 31-Aug-2000, PCT Appln. No.US00/26013 filed on 22-Sep-2000, PCT Appln. No.US00/28664 filed on 17-Oct-2000, US Appln. No. 09/833,245 15 filed on 12-Apr-2001, and PCT Appln. No. US01/11988 filed on 12-Apr-2001.

**International Application** Applicant's File Unassigned PS907PCT Reference Number: Number:

# INDICATIONS RELATING TO DEPOSITED BIOLOGICAL MATERIAL

(PCT Rule 13bis)

A. The indications made below relate to the deposited biological material referred to in Table 1A of the description.

B. <u>IDENTIFICATION OF DEPOSIT</u>:

Further deposits are identified on an additional sheet:

X

Name of Depository:

**American Type Culture Collection** Address of Depository: 10801 University Boulevard Manassas, Virginia 20110-2209 United States of America

	Accession	Date of	1 1	Accession	Date of
	Number	Deposit		Number	Deposit
1	203027	26-Jun-1998	2	209423	30-Oct-1997
3	203069	27-Jul-1998	4	209463	14-Nov-1997
5	203070	27-Jul-1998	6	209511	3-Dec-1997
7	203071	27-Jul-1998	8	209551	12-Dec-1997
9	203081	30-Jul-1998	10	209563	18-Dec-1997
11	203105	13-Aug-1998	12	209568	6-Jan-1998
13	203181	9-Sep-1998	14	209580	14-Jan-1998
15	203331	8-Oct-1998	16	209603	29-Jan-1998
17	203364	19-Oct-1998	18	209626	12-Feb-1998
19	203499	1-Dec-1998	20	209627	12-Feb-1998
21	203517	10-Dec-1998	22	209628	12-Feb-1998
23	203570	11-Jan-1999	24	209641	25-Feb-1998
25	203648	9-Feb-1999	26	209651	4-Mar-1998
27	203858	18-Mar-1999	28	209683	20-Mar-1998
29	209007	28-Apr-1997	30	209745	7-Apr-1998
31	209008	28-Apr-1997	32	209746	7-Apr-1998
33	209009	28-Apr-1997	34	209782	20-Apr-1998
35	209010	28-Apr-1997	36	209852	7-May-1998
37	209011	28-Apr-1997	38	209853	7-May-1998
39	209012	28-Apr-1997	40	209877	18-May-1998
41	209022	8-May-1997	- 42	209878	18-May-1998
43	209045	15-May-1997	44	209889	22-May-1998
45	209070	22-May-1997	46	209965	11-Jun-1998
47	209071	22-May-1997	48	97899	26-Feb-1997
49	209072	22-May-1997	50	97922	7-Mar-1997
51	209073	22-May-1997	52	97923	7-Mar-1997
53	209074	22-May-1997	54	97955	13-Mar-1997
55	209075	22-May-1997	56	97957	13-Mar-1997
57	209080	29-May-1997	58	97958	13-Mar-1997
59	209081	29-May-1997	60	97974	4-Apr-1997
61	209082	29-May-1997	62	97975	4-Apr-1997
63	209083	29-May-1997	64	97976	4-Apr-1997

Applicant's File		International Application		
Reference Number:	PS907PCT	Number:	Unassigned	

	Accession	Date of		Accession	Date of
	Number	Deposit		Number	Deposit
65	209084	29-May-1997	66	97977	4-Apr-1997
67	209085	29-May-1997	68	97978	27-Mar-1997
69	209086	29-May-1997	70	97979	27-Mar-1997
71	209089	5-Jun-1997	72	PTA-1543	21-Mar-2000
73	209090	5-Jun-1997	74	PTA-1544	21-Mar-2000
75	209119	12-Jun-1997	76	PTA-163	1-Jun-1999
77	209124	19-Jun-1997	78	PTA-2069	9-Jun-2000
79	209125	19-Jun-1997	80	PTA-2070	9-Jun-2000
81	209126	19-Jun-1997	82	PTA-2075	9-Jun-2000
83	209138	3-Jul-1997	84	PTA-2076	9-Jun-2000
85	209139	3-Jul-1997	86	PTA-2081	9-Jun-2000
87	209145	17-Jul-1997	88	PTA-322	9-Jul-1999
89	209146	17-Jul-1997	90	PTA-499	11-Aug-1999
91	209147	17-Jul-1997	92	PTA-622	2-Sep-1999
93	209179	24-Jul-1997	94	PTA-623	2-Sep-1999
95	209180	24-Jul-1997	96	PTA-841	13-Oct-1999
97	209195	1-Aug-1997	98	PTA-842	13-Oct-1999
99	209215	21-Aug-1997	100	PTA-843	13-Oct-1999
101	209224	28-Aug-1997	102	PTA-844	13-Oct-1999
103	209225	28-Aug-1997	104	PTA-845	13-Oct-1999
105	209236	4-Sep-1997	106	PTA-846	13-Oct-1999
107	209241	12-Sep-1997	108	PTA-847	13-Oct-1999
109	209242	12-Sep-1997	110	PTA-848	13-Oct-1999
111	209243	12-Sep-1997	112	PTA-849	13-Oct-1999
113	209244	12-Sep-1997	114	PTA-855	18-Oct-1999
115	209277	18-Sep-1997	116	PTA-867	26-Oct-1999
117	209299	25-Sep-1997	118	PTA-868	26-Oct-1999
119	209300	25-Sep-1997	120	PTA-869	26-Oct-1999
121	209324	2-Oct-1997	122	PTA-871	26-Oct-1999
123	209346	9-Oct-1997	124	PTA-883	28-Oct-1999
125	209368	16-Oct-1997	126	PTA-884	28-Oct-1999
127	209407	23-Oct-1997	128	PTA-885	28-Oct-1999

## **EUROPE**

In respect of those designations in which a European Patent is sought a sample of the deposited microorganism will be made available until the publication of the mention of the grant of the European patent or until the date on which the application has been refused or withdrawn or is deemed to be withdrawn, only by the issue of such a sample to an expert nominated by the person requesting the sample (Rule 28(4) EPC).

# **CANADA**

The applicant requests that, until either a Canadian patent has been issued on the basis of an application or the application has been refused, or is abandoned and no longer subject to reinstatement, or is withdrawn, the Commissioner of Patents only authorizes the furnishing of a sample of the deposited biological material referred to in the application to an independent expert nominated by the Commissioner, the applicant must, by a written statement, inform the International Bureau accordingly before completion of technical preparations for publication of the international application.

### **NORWAY**

The applicant hereby requests that the application has been laid open to public inspection (by the Norwegian Patent Office), or has been finally decided upon by the Norwegian Patent Office without having been laid open inspection, the furnishing of a sample shall only be effected to an expert in the art. The request to this effect shall be filed by the applicant with the Norwegian Patent Office not later than at the time when the application is made available to the public under Sections 22 and 33(3) of the Norwegian Patents Act. If such a request has been filed by the applicant, any request made by a third party for the furnishing of a sample shall indicate the expert to be used. That expert may be any person entered on the list of recognized experts drawn up by the Norwegian Patent Office or any person approved by the applicant in the individual case.

### **AUSTRALIA**

The applicant hereby gives notice that the furnishing of a sample of a microorganism shall only be effected prior to the grant of a patent, or prior to the lapsing, refusal or withdrawal of the application, to a person who is a skilled addressee without an interest in the invention (Regulation 3.25(3) of the Australian Patents Regulations).

## **FINLAND**

The applicant hereby requests that, until the application has been laid open to public inspection (by the National Board of Patents and Regulations), or has been finally decided upon by the National Board of Patents and Registration without having been laid open to public inspection, the furnishing of a sample shall only be effected to an expert in the art.

### UNITED KINGDOM

The applicant hereby requests that the furnishing of a sample of a microorganism shall only be made available to an expert. The request to this effect must be filed by the applicant with the International Bureau before the completion of the technical preparations for the international publication of the application.

## **DENMARK**

The applicant hereby requests that, until the application has been laid open to public inspection (by the Danish Patent Office), or has been finally decided upon by the Danish Patent office without having been laid open to public inspection, the furnishing of a sample shall only be effected to an expert in the art. The request to this effect shall be filed by the applicant with the Danish Patent Office not later that at the time when the application is made available to the public under Sections 22 and 33(3) of the Danish Patents Act. If such a request has been filed by the applicant, any request made by a third party for the furnishing of a sample shall indicate the expert to be used. That expert may be any person entered on a list of recognized experts drawn up by the Danish Patent Office or any person by the applicant in the individual case.

#### **SWEDEN**

The applicant hereby requests that, until the application has been laid open to public inspection (by the Swedish Patent Office), or has been finally decided upon by the Swedish Patent Office without having been laid open to public inspection, the furnishing of a sample shall only be effected to an expert in the art. The request to this effect shall be filed by the applicant with the International Bureau before the expiration of 16 months from the priority date (preferably on the Form PCT/RO/134 reproduced in annex Z of Volume I of the PCT Applicant's Guide). If such a request has been filed by the applicant any request made by a third party for the furnishing of a sample shall indicate the expert to be used. That expert may be any person entered on a list of recognized experts drawn up by the Swedish Patent Office or any person approved by a applicant in the individual case.

#### **NETHERLANDS**

The applicant hereby requests that until the date of a grant of a Netherlands patent or until the date on which the application is refused or withdrawn or lapsed, the microorganism shall be made available as provided in the 31F(1) of the Patent Rules only by the issue of a sample to an expert. The request to this effect must be furnished by the applicant with the Netherlands Industrial Property Office before the date on which the application is made available to the public under Section 22C or Section 25 of the Patents Act of the Kingdom of the Netherlands, whichever of the two dates occurs earlier.

## What Is Claimed Is:

1. Use of a polypeptide for the preparation of a diagnostic or pharmaceutical composition for diagnosing or treating a hematopoietic or hematologic disorder, wherein said polypeptide comprises an amino acid sequence at least 95% identical to a sequence selected from the group consisting of:

- (a) a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A:
- (b) a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (d) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;
  - (e) a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
  - (f) a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
  - (g) a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 2. Use of the polypeptide of claim 1, wherein said wherein said polypeptide comprises a heterologous amino acid sequence.
- 3. Use of a polypeptide for the preparation of a diagnostic or pharmaceutical composition for diagnosing or treating a hematopoietic or hematologic disorder, wherein said polypeptide comprises an amino acid sequence selected from the group consisting of:
- (a) a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (b) a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;

(c) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;

- (d) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;
  - (e) a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
  - (f) a polypeptide domain of SEQ ID NO: Y as referenced in Table 2; and
  - (g) a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 4. Use of the polypeptide of claim 3, wherein said polypeptide comprises a heterologous amino acid sequence.
- 5. Use of an antibody or fragment thereof for the preparation of a diagnostic or pharmaceutical composition for diagnosing or treating a hematopoietic or hematologic disorder, wherein said antibody or fragment thereof binds a polypeptide comprising an amino acid sequence at least 95% identical to a sequence selected from the group consisting of:
- (a) a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (b) a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A:
- (d) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;
  - (e) a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
  - (f) a polypeptide domain of SEQ ID NO: Y as referenced in Table 2; and
  - (g) a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- Use of an antibody or fragment thereof for the preparation of a diagnostic or pharmaceutical composition for diagnosing or treating a hematopoietic or hematologic disorder,

wherein said antibody or fragment thereof binds a polypeptide selected from the group consisting of:

- (a) a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (b) a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A:
- (c) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (d) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;
  - (e) a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
  - (f) a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
  - (g) a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 7. Use of a nucleic acid molecule for the preparation of a diagnostic or pharmaceutical composition for diagnosing or treating a hematopoietic or hematologic disorder, wherein said nucleic acid molecule comprises a polynucleotide sequence at least 95% identical to a sequence selected from the group consisting of:
  - (a) a polynucleotide fragment of SEQ ID NO:X as referenced in Table 1A;
- (b) a polynucleotide encoding a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polynucleotide encoding a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (d) a polynucleotide encoding a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (e) a polynucleotide encoding a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;

(f) a polynucleotide encoding a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;

- (g) a polynucleotide encoding a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
- (h) a polynucleotide encoding a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 8. Use of the nucleic acid molecule of claim 7, wherein said nucleic acid molecule comprises a heterologous polynucleotide sequence.
- 9. Use of a nucleic acid molecule for the preparation of a diagnostic or pharmaceutical composition for diagnosing or treating a hematopoietic or hematologic disorder, wherein said nucleic acid molecule comprises a polynucleotide sequence selected from the group consisting of:
  - (a) a polynucleotide fragment of SEQ ID NO:X as referenced in Table 1A;
- (b) a polynucleotide encoding a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polynucleotide encoding a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEO ID NO:Y as referenced in Table 1A;
- (d) a polynucleotide encoding a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEO ID NO:Y as referenced in Table 1A;
- (e) a polynucleotide encoding a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;
- (f) a polynucleotide encoding a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B:
- (g) a polynucleotide encoding a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
- (h) a polynucleotide encoding a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 10. Use of the nucleic acid molecule of claim 9, wherein said nucleic acid molecule comprises a heterologous polynucleotide sequence.

11. Use of an agonist or antagonist for the preparation of a pharmaceutical composition for treating a hematopoietic or hematologic disorder, wherein said agonist or antagonist binds a polypeptide comprising an amino acid sequence at least 95% identical to a sequence selected from the group consisting of:

- (a) a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (b) a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (d) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;
  - (e) a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
  - (f) a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
  - (g) a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 12. Use of an agonist or antagonist for the preparation of a pharmaceutical composition for treating a hematopoietic or hematologic disorder, wherein said agonist or antagonist binds a polypeptide selected from the group consisting of:
- (a) a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (b) a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;

(d) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;

- (e) a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
- (f) a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
- (g) a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 13. A polypeptide comprising an amino acid sequence at least 95% identical to a sequence selected from the group consisting of:
- (a) a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (b) a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (d) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;
  - (e) a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
  - (f) a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
  - (g) a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 14. The polypeptide of claim 13, wherein said polypeptide comprises a heterologous amino acid sequence.
  - 15. Use of the polypeptide of claim 13 for identifying a binding partner comprising:
    - (a) contacting the polypeptide of claim 13 with a binding partner; and
- (b) determining whether the binding partner increases or decreases activity of the polypeptide.
- 16. A polypeptide comprising an amino acid sequence selected from the group consisting of:

(a) a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;

- (b) a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (d) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;
  - (e) a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
  - (f) a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
  - (g) a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 17. The polypeptide of claim 16, wherein said polypeptide comprises a heterologous polypeptide sequence.
  - 18. Use of the polypeptide of claim 16 for identifying a binding partner comprising:
    - (a) contacting the polypeptide of claim 16 with a binding partner; and
- (b) determining whether the binding partner increases or decreases activity of the polypeptide.
- 19. An antibody or fragment thereof that binds a polypeptide comprising an amino acid sequence at least 95% identical to a sequence selected from the group consisting of:
- (a) a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (b) a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;

(d) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;

- (e) a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
- (f) a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
- (g) a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 20.An antibody or fragment thereof that binds a polypeptide selected from the group consisting of:
- (a) a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (b) a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (d) a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;
  - (e) a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
  - (f) a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
  - (g) a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 21. A nucleic acid molecule comprising a polynucleotide sequence at least 95% identical to a sequence selected from the group consisting of:
  - (a) a polynucleotide fragment of SEQ ID NO:X as referenced in Table 1A;
- (b) a polynucleotide encoding a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polynucleotide encoding a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;

(d) a polynucleotide encoding a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;

- (e) a polynucleotide encoding a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;
- (f) a polynucleotide encoding a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
- (g) a polynucleotide encoding a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
- (h) a polynucleotide encoding a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 22. The nucleic acid molecule of claim 21, wherein said nucleic acid molecule comprises a heterologous polynucleotide sequence.
  - 23. A recombinant vector comprising the nucleic acid molecule of claim 21.
  - 24. A recombinant vector comprising the nucleic acid molecule of claim 22.
  - 25. A recombinant host cell comprising the recombinant vector of claim 23.
  - 26. A recombinant host cell comprising the recombinant vector of claim 24.
- 27. A nucleic acid molecule comprising a polynucleotide sequence selected from the group consisting of:
  - (a) a polynucleotide fragment of SEQ ID NO:X as referenced in Table 1A;
- (b) a polynucleotide encoding a full length polypeptide of SEQ ID NO:Y or a full length polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;
- (c) a polynucleotide encoding a predicted secreted form of SEQ ID NO:Y or a secreted form of the polypeptide encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;

(d) a polynucleotide encoding a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEQ ID NO:Y as referenced in Table 1A;

- (e) a polynucleotide encoding a polypeptide fragment of SEQ ID NO:Y or a polypeptide fragment encoded by the cDNA Clone ID in ATCC Deposit No:Z corresponding to SEO ID NO:Y as referenced in Table 1A, wherein said fragment has biological activity;
- (f) a polynucleotide encoding a polypeptide domain of SEQ ID NO:Y as referenced in Table 1B;
- (g) a polynucleotide encoding a polypeptide domain of SEQ ID NO:Y as referenced in Table 2; and
- (h) a polynucleotide encoding a predicted epitope of SEQ ID NO:Y as referenced in Table 1B.
- 28. The nucleic acid molecule of claim 27, wherein said nucleic acid molecule comprises a heterologous polynucleotide sequence.
  - 29. A recombinant vector comprising the nucleic acid molecule of claim 27.
  - 30. A recombinant vector comprising the nucleic acid molecule of claim 28.
  - 31. A recombinant host cell comprising the recombinant vector of claim 29.
  - 32. A recombinant host cell comprising the recombinant vector of claim 30.